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Influence of Tomato Powder on Growth Performance and Health State in Broilers Diet Exposed to Aflatoxin

Thesis

Submitted to the council of the College of Veterinary Medicine at University of Kerbala as a Partial fulfillment of the Requirement for the Degree of Master in the Sciences of Veterinary Medicine in Veterinary Public Health

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بسم ٱللّٰه الرَّحْمٰنِ الرَّحِيـمِ

﴿ وَالْأَنْعَامَ خَلَقَهَا ۗ لَكُمْ فِيهَا دِفْءٌ وَمَنَافِعُ وَمِنْهَا تَأْكُلُونَ ﴾

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I hereby declare that this thesis is my origin work except for equations and citations which have been fully acknowledged. I also declare that it has not been previously, and is not concurrently, submitted for any other degree at University of Kerbala or other institutions.

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Dedication

To almighty Allah Creator of the heavens and the earth.

To my wounded homeland…. IRAQ

I present fragments of my humble research as a gift to my master, the Awaited Alimam Mahdi (may God bless him and grant him peace).

For those who strive to console me and make me happy.... My father

For whom is that paradise under her feet...My mother

To my wife who supported me and encouraged me in every step and my beloved daughter Mina

To my dears... my brothers and sisters

To my friends and colleagues in my studies and all my teachers, to Hussein Adnan

To my companion in my path, who stood with me in all the steps of my search, my cousin Maher

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I would like to thank everyone who supported me to successfully complete my research

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Amjad Abdul-Hassan Al-Dawoodi

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List of abbreviations

Abbreviations	Full wordS
AF	Aflatoxin
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
BW	Body Weight
Con	Control
ELISA	Enzyme Linked Immunosorbent Assay
FAV	Fruits and vegetables
FCR	Feed conversion ratio
FI	Feed intake
Gpx	Glutathione Peroxidase
HDL	High density lipoprotein
HPLC	High performance liquid chromatographic
IBD	Infectious Bursal Disease
LDL	Low density lipoprotein
LYC	Lycopene
M	Mycofix
MDA	Malondialdehyde
ND	Newcastle Disease
ppb	Part per billion
SOD	Superoxide dismutase
TP	Tomato powder
VLDL	Very low density lipoprotein

WG	Weight gain

Abstract

The present study was conducted at College of Veterinary Medicine / University of Kerbala to investigate the effect of the Tomato powder in reducing the damage caused by the exposure of broiler chickens to aflatoxins by measuring some biochemical, antioxidant, immunological, performance, and histological changes of liver in broilers.

The experiment lasted for 35 days from 12 Feb to 19 Mar 2022. One hundred broiler chicks (Ross 308) at one day aged were randomly divided into 5 equal groups (20 chicks per group). The first group was considered a general control group fed on a basal diet free from any additions. The second group fed the aflatoxin (AF) contaminated diet daily by 30 ppb. The third group fed on a diet contaminated with aflatoxin 30 ppb+ supplemented by mycofix concentration 0.1 g / kg feed. The fourth group was also fed on a diet contaminated with aflatoxin 30 ppb+ supplemented by tomato powder (TP) with concentration 10 g / kg feed. The fifth group fed on a basal diet supplemented with Tomato powder with concentration 10 g / kg feed during the experiment. Powder of tomato components were analyzed by high performance liquid chromatographic (HPLC) for its components.

At day 4th, 28th and 35th of their age, blood samples were collected from 5 bird per group from the jugular vein in a test tube without anticoagulant, to obtain the serum to determine the biochemical and immunological tests.

Results of the biochemical parameters of the current study revealed that the AF&TP group showed a significant decrease ($P<0.05$) in the concentration of Aspartate aminotransferase (AST) and Alanine aminotransferase (ALT), as well as the level of creatinine compared with AF group, which showed a significant increase ($P<0.05$) in the concentration of the same parameters in day 28, while in day 35 showed a significant decrease ($P<0.05$) in the concentration the level of creatinine compared with AF group.

The levels of cholesterol, triglyceride, low density lipoprotein (LDL) and very low density lipoprotein (VLDL) were significantly decrease (P <0.05) in the AF&TP group compared to the AF group that showed a significant increased (P<0.05). Also high density lipoprotein (HDL) were significantly increase (P <0.05) in the AF&TP group compared to the AF group that showed a significant decrease (P<0.05) in day 28 and 35. As well, the results showed significant increase (P<0.05) in the concentration of glutathione peroxidase (Gpx) and superoxide dismutase (SOD) in the AF&TP group compared to the AF group. In addition, the results of malondialdehyde (MDA) significantly decreased (P<0.05) in the AF&TP group compared to the AF group in day 28, while in day 35 showed a significant increase (P<0.05) in the concentration of Gpx in the AF&TP group compared to the AF group.

The sensory traits results showed a significant increase (P<0.05) in the tenderness, juicy, flavor, color and palatability in the AF&TP group compared to the AF group. The results that showed a significant increase (P<0.05) in the weight of spleen, thymus and bursa of fabricius in the AF&TP group compared to the AF group, in addition, the results as a significant decrease (P<0.05) in the weight of liver in the AF&TP group compared to the AF group in day 28, while in day 35 showed a significant decrease (P<0.05) in the weight of liver in the AF&TP group compared to the AF group. The result of the hepatosomatic index showed a significant decrease (P<0.05) in the AF&TP group compared to the AF group. The immunological results showed a significant increase (P<0.05) in the levels of antibodies against Newcastle disease and infectious bursal disease for the AF&TP group compared with the AF group, which showed a significant decrease (P<0.05) in the same parameters. The results of the body weight in day 7,14,21 and 28 showed a significant increase (P<0.05) in the AF&TP group compared to the AF group for the same results.

Histopathological changes revealed improvement in the damaged tissue of the AF&TP group compared to the AF group by reducing the hemorrhage and the return of tissue relative to the normal structure. Its concluded from the results of this study that, the tomato powder at 10 g/kg has a protective role to reduce the damage that caused by exposure to 30 ppb aflatoxin in feed of broiler.

Chapter One: Introduction

Introduction

Chicken meat is an important source of nutrients for human consumption, its more superior to red meat due to several other reasons, including its health benefits, as it contains less fat and cholesterol, easy to handle portions and less religious barriers, few fast-growing commercial broiler strains play the vital role in producing the required amount of chicken meat to the world population (Jayasena *et al*., 2013). Broiler feed it exposed to various contaminants during the process of production, transportation and storage. Moisture is the most important factor favoring the growth of fungus during these stages (Kralik *et al*., 2018). Emphasis is put on importance of chicken meat consumption for maintaining body weight due to the intake of dietary protein. Chicken meat is often a part of the diet aimed to reduce body weight, because of its high protein and low fat content (Te Morenga& Mann, 2012 ; Astrup *et al*., 2015).

Mycotoxins, the toxic secondary metabolites of fungi, particularly produced by many species of *Aspergillus*, *Fusarium* and *Penicillium*, have affected animal and human health for over thousand years, whereas little has been discovered so far about these complex substances in poultry, which are generally very sensitive. The most common effects are reduced feed intake, weight gain, feed efficiency, growth performance, immunity and hatchability, organ damages (mainly kidney and liver), carcinogenicity, teratogenicity, decreased egg production and increased mortality (Filazi *et al*., 2017). Among these mycotoxins are AF, ochratoxin A , zearalenone, T-2 toxin, vomitoxin, and fumonisin (Aravind *et al*., 2003).

Aflatoxins are one of the most widespread pollutants in poultry feed in both developing and developed countries (Rushing & Selim 2019 ; Hernández-Ramírez *et al*.,2021). At present, many AF have been identified, AF is the most toxic for chicken and has been classified as a class I carcinogen by the International Agency for Research on Cancer (Elwan *et al*., 2021; Li *et al*., 2021). AF is often found in corn and peanut, which is the major energy sources for the poultry feed, the permitted level of AF is very low in poultry feed, and thus poultry feed is at a high risk of contamination with AF (Fouad *et al*., 2019). It has been reported that the most vulnerable body organ of chicken to AF is the liver (Li *et al*., 2021). AF can induce unbalanced lipid metabolism, inhibit the activity of antioxidant enzymes, increase pro-inflammatory cytokines and increase hepatocyte apoptosis levels (Rosa *et al*., 2001; Fouad *et al*., 2019 ; Li *et al*.,

2021). In addition, dietary exposure to AF is associated with immune dysfunction of chickens, making the broilers more susceptible to infectious diseases (Qureshi *et al*., 1998 ; Shivachandra *et al*., 2003). It has been reported that AF could induce apoptosis in immune organs, and cause significant decrease in the production of immunoglobulin (Fouad *et al*., 2019).

Tomato is a well-studied species belonging to the Solanaceae family. Its consumption, as well as production, is increasing because of its antioxidant and anti-cancerous properties (Rai *et al*., 2021). Tomato components like LYC, phenolic, flavonoids, vitamins C and E mainly have the antioxidant capacity and processed tomato products (Beutner *et al*., 2001). Tomato powder is high in lycopene is a natural food-derived pigment belonging to carotenoids used in food processing, and is mainly enriched in fruits and vegetables with a red color (Liang *et al*., 2019). LYC can be used as a bioactive plant food material with many vital activities, including antioxidant capacity, and has therapeutic potential against diseases (Grabowska *et al*., 2019).

Aim of Study: -

Due to the importance of poultry production, the purpose of the study is to show the efficacy of Tomato powder in alleviating the deleterious effects of aflatoxin in broilers, by the following parameters

1. Some biochemical serum ALT, AST, Determination of oxidative study through MDA, (SOD), (Gpx) **,**Blood lipid profiles (cholesterol, Triglcyride, HDL, LDL, VLDL).

2. The immunological status by ELISA for ND & IBD.

3. The body weight performance (body weight, body weight gain, Feed intake, Feed conversion ratio will be checked on day 7, 14, 21, 28 and 35) and Hepatosomatic index

4. Histopathological changes of the liver (at day 28).

Chapter Two: Review Of The Related Literatures

2.Literatures Review

2.1. Poultry production

Historically, the poultry sector has evolved through three phases: i) traditional systems, which include family poultry consisting of scavenging birds and backyard raising; ii) small-scale semi-commercial systems; and iii) large-scale commercial systems. Each of these systems is based on a unique set of technologies. They differ markedly in investment, type of birds used, husbandry level and inputs such as feeds. The feed resources, feeding and feed requirements required to raise poultry also vary widely, depending on the system used (Ravindran, 2013).

Chicken meat is an important source of nutrients for human consumption. Broiler feed it exposed to various contaminants during the process of production, transportation and storage. Moisture is the most important factor favoring the growth of fungus during these stages (Chand *et al*., 2011). A proprietary toxin deactivator (Mycofix®) containing Trichosporon mycotoxinivorans was used to assess its presumed protection against the toxic effects of mycotoxin on broiler chicken (Hanif *et al*., 2008).

Chickens are kept in a wide range of production systems, and provide mainly meat, eggs and manure for crop fertilization. Poultry meat and eggs are among the most common animal source food consumed at the global level, through a wide diversity of cultures, traditions and religions, making them key to food security and nutrition. Within the livestock sector, poultry emerges as the most efficient sub-sector in its use of natural resources and providing protein to supply a global growing demand. Poultry is particularly important for small holders and poor rural and urban communities and is mainly produced in large scale and intensive operations, making it one of the fastest growing agricultural sub-sectors, while most of the sector's growth has been driven by private investments, public concerns about the sector's impact on the environment and human health, its contribution to climate change and to local and global economy is triggering governments' response and the development of public policies for the sector (Mottet & Tempio, 2017).

Chicken meat consumption for maintaining and reducing body weight. It is known that the intake of dietary protein is effective in reducing body weight, because of its high protein and low fat content (Te Morenga & Mann, 2012 ; Astrup *et al*., 2015).

Chicken meat is considered as desirable foodstuff in prevention of cardiovascular diseases (Abete *et al*., 2014). According to the data of (Bernstein *et al*., 2010) by replacing meals with red meat with white chicken meat, the risk of cardiovascular disease occurrence can be lowered by 19%.

2.2. Mycotoxins

There are various mycotoxis in the evolved way of life that reason undesirable natural impacts inside human and creature life forms upon ingestion (Bryden, 2007). Abnormal state of mycotoxins in nourishment and feed brings about the presence of intense mycotoxicosis and high death rate. Lower levels cause the event of unending mycotoxicosis with or without showing clinical side effects. However, followed by a considerable decrease in production performance, immunosuppressive impacts, and the event of deposits in poultry meat and eggs (Resanovic & Sinovec, 2006).

It is not astounding in this way, that mycotoxins are the reason for various distinctive creature sicknesses and those subsequent from ingestion of grain-based eating regimens. The level of poisonous quality will rely upon the toxin introduce, measurements, span of presentation, and an assortment of difference variables. Species, age, hormonal status, sustenance and simultaneous ailment are viewed as the most important (Bryden, 2007; Wild, 2007). The gut microflora may likewise adjust mycotoxin poisonous quality (Swanson *et al*., 1988). The fungal toxicity comes from the ability of these toxins to cause changes and mutations on DNAas a result of these changes possible innate toxins are carcinogenic (e.g. fumonisins), carcinogenic and teratogenic (e.g. ochratoxin A), or carcinogenic, mutagenic and teratogenic (e.g., aflatoxin) (Cawdell-Smith *et al*., 2007).

2.3. Aflatoxins (AF)

Aflatoxins are the naturally occurring mycotoxins, produced as secondary metabolites by the fungus *Aspergillus flavus, A. parasiticus,* and *A. nominus*. The name ‘‘aflatoxin” is derived from the first letter in *Aspergillus*, and the first three letters in *flavus* (Schoental, 1967 ; Marin *et al*., 2019). The carcinogenicity and high toxicity for animals and humans possessed so that aflatoxin has made it the subject of many researchers. Poultry is resistant to aflatoxin to some extent because it is acute and requires weeks of exposure to a chronic injury (one week minimum). The risk of

aflatoxin toxicity lies in its ability to penetrate the cell and nucleus and interfere between the constituent bases of the DNA, leading to malfunction and slow transfer and replication of DNA, which result in the inhibition of protein synthesis, i.e. “wrong" proteins are synthesized. Although the mechanism of immunosuppression was not fully explained, the role of aflatoxin was determined to damage the completed proteins, interferons, and blood serum, which were due to damage to the liver and inhibition of protein synthesis. Aflatoxin performs suppression of nonspecific substances (complement and interferon) in charge of humoral immunity, as well as the suppression of phagocytes through macrophages (Murarolli, 2013).

Structurally, AFs are difurocoumarin derivatives that fluoresce under ultraviolet light. Depending upon color of the fluorescence, AFs are divided into aflatoxin B1 and B2 (AFB1, AFB2) for blue, and G1 and G2 (AFG1, AFG2) for green (Dalvi, 1986) (figure 2-1). Aflatoxin M1 and M2 (AFM1, AFM2), Other metabolites of AFB1 are aflatoxin Q1 (AFQ1) and aflatoxicol. Aflatoxins are the most intensively researched group of mycotoxins, because of their demonstrated toxic and carcinogenic effects in the susceptible laboratory animals and livestock and their acute toxicological and chronic hepatocarcinogenic effects in humans (Bondy & Pestka, 2000).

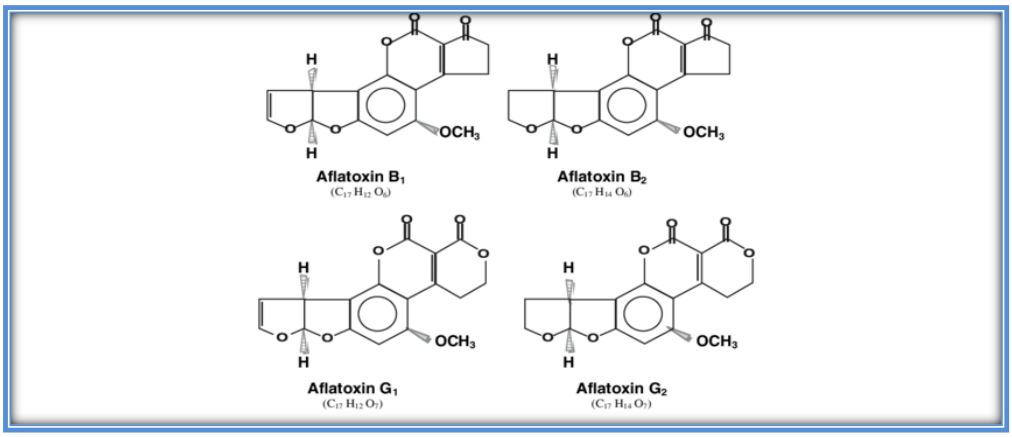

Figure (2-1) structure of aflatoxin types (Dalvi, 1986)

2.3.1. Biosynthesis of aflatoxins and their metabolites

Biosynthesis of aflatoxins involves polyketide pathways having very complicated process in contribution with more than 25 clusters of genes positioned in a 75 kb region of fungal chromosome (Abyaneh *et al*., 2008). It involves many enzymes and genes. However, a large variety of genera and species produce the anthraquinones and some of them produce the xanthones. Final step of oxidative ring cleavage is proceeded by some tropical *Aspergilli* and results in the synthesis of aflatoxin from xanthones. Even among the *Aspergilli*, *Aspergillus flavus* has ability to produce only B group, while *Aspergillus parasiticus* can do more oxidative ring expansion and can produce G group (Moss, 2002).

2.3.2. Occurrence of Aflatoxins

Aflatoxins contaminate food and feed during growth, harvest and storage. Aflatoxins are also present in peanuts, milk, corn, cottonseed, almonds, nuts, spices, cheese and a range of other feed and food products. Aflatoxin can be present in eggs, milk and meat products, as the animals also ingest the aflatoxin-infected feed. Although, the food product such as peanuts, corn and cottonseed has the highest risk of aflatoxin contamination (Colak *et al*., 2006).

2.3.3. Factors Favorizing Aflatoxin Production

Several factors are responsible for aflatoxin production. Aflatoxin production is the outcome of relationship among the host, fungus and the environment. The suitable interactions of these factors are responsible for the type and concentration of aflatoxin produced. Major causes in mold infestation and toxin production are high-temperature stress, humidity (16%) and factors which decrease immunity of host such as insect damage (Roger & Coulombe, 1993). In plants, such as maize and peanuts, the spores of *Aspergillus paraciticus* and *Aspergillus flavus* can grow on the surface of stigma. The germ tube goes into the developing embryo and mimics pollen germ tubes. So, the mycelium's develop an end trophic interaction which does not damage the healthy plant. But if the plant is stressed than aflatoxins can be produced in the plant tissue during the development in the field and the most common stress is drought. Food can also be contaminated during harvesting but the concentrations of aflatoxins are not as high as in stored food products. Animal feeds, milk and milk products can be contaminated by aflatoxins even in temperate climates where these Aspergilli do not

generally participate. However, their spores are present in the air all over the world (Moss, 2002) and can be dispersed easily from their natural ecological niches to the vulnerable crops and plants throughout the world (Abyaneh *et al*., 2008).

2.3.4. Exposure to Aflatoxins

Aflatoxicosis poisoning that occurs from ingesting aflatoxins, is characterized in broiler chickens by decreased feed intake and growth rate, poor feed utilization, and mortality. The productive deterioration is also associated with changes in biochemical and hematological parameters, liver and kidney abnormalities, and impaired immunity, its able to enhance susceptibility to infectious diseases. Thus, aflatoxicosis causes severe economic losses in the poultry industry. Furthermore, the transmission of AF and its metabolites from feed to animal edible tissues and products, such as liver and eggs becomes particularly important as apotential hazard for human health (Denli *et al*., 2009).

Humans are mostly infected by direct ingestion of infected crops (Egal *et al*., 2005), by eating foods (oil, peanut butter, wine, nut paste, etc) derived from infected commodities (Colak *et al*., 2006). Aflatoxins can also be ingested through infected milk containing AFM1 (Hussain *et al*., 2008).

2.3.5. Aflatoxin toxicity in poultry

Poultry are extremely sensitive to the toxic effects of AF (Arafa *et al*., 1981; Klein *et al*., 2000). The disease was shown to be caused by AF contaminated feed, among difference poultry species. European community regulatory limit of 20 μg/kg and the Food and Agriculture Organization (FAO)/World Health Organization (WHO) recommended maximum permissible limit of 30 μg/kg (30 ppb) for poultry feeds. (Aboagye-Nuamah *et al*., 2021), poultry, known to be extremely sensitive to AF, responded to doses as low as 15–30 μg/kg and the levels needed for equivalent pathology were as high as 100 μg/kg for those from chickens (Rawal *et al*., 2010).

2.3.6. Effect Aflatoxins on immune system

Aflatoxins work as an inhibitor of protein synthesis and as resulted destroyed cells and tissues with a high protein turnover such as that found in the liver, immune system or gut epithelium, which is most susceptible to the toxic effects of AF. In this way, exposure to AF has been demonstrated to suppress the immune response in poultry.AF can inhibits the development of the thymus gland or effect the relative weight of the bursa of Fabricius, which may result in serious deficiencies in both cellular and antibody responsiveness of the chicken immune system (Celik *et al*, 2000). Inhibition of macrophage functions, T lymphocyte activity or cytokine expression by AF results in vaccine failure or pathogen persistence, as exemplified in many studies by reduced immunoglobulin production (Yunus *et al*, 2008). Recent epidemiological data indicates a high correlation between outbreak of Newcastle disease and AF contamination of broiler rations (Yunus *et al*., 2008).

2.4. Oxidative stress

It's an imbalance between the production of reactive oxygen species (ROS) in the body that disrupts its ability to detoxify reactive intermediates, or to repair the damage to organ and cellular systems that can be caused by ROS. Intracellular redox* balance (the dynamic balance between oxidizing and reducing species within cells) is closely coupled to the antioxidant peptide glutathione; intracellular levels of this compound are highly regulated by enzymes to maintain a reducing environment. Some of these reactive oxygen species are constantly produced at low levels as by-products of normal metabolic reactions, and are kept in check by the enzymes that maintain the intracellular redox balance. When redox balance is upset, these moderately reactive species can interact with transition metals or other components of the redox cycle to produce highly reactive oxygen species that can cause extensive damage to cell membranes lipids, proteins, DNA, and cellular organelles (Jones& Sies, 2007).

2.4.1.Oxidative stress of aflatoxin

Aflatoxin can cause an increase in ROS formation in animals' target organs, including rat liver, duck liver, and mouse lung. AF induced an important liver cell injury, as shown by the significant increase in serum transaminases, phosphatases (acid and alkaline), dehydrogenases (sorbitol, lactate and glutamate), cholesterol, triglycerides, total lipids, bilirubin, creatinine, uric acid and nitric oxide, but also a

strong lipid peroxidation in liver and kidney, accompanied with a significant decrease in total antioxidant capacity. Recently, its accepted that oxidative stress is an apoptosis inducer, many agents that induce apoptosis are either oxidants or stimulators of cellular oxidative metabolism (Marin & Taranu, 2012).

2.5. Medicinal plant

These are products which are obtained from plant resources for the treatment and wellbeing of mankind and animals (Balekundri & Mannur, 2020).

Various herbs play important role in production and reproduction of animal. Herbs acts as feed additives, growth promoters, immune boosters, improves reproduction in animals (Kuralkar & Kuralkar, 2021).

Among the various food plants, fruits and vegetables (FAV) are reported to have health-improving benefits. Some FAV such as the citrus fruits (orange, grapefruit, lime, lemon), tomato, grapes, pomegranates, apples, dates, green and yellow vegetables (peppers), cabbage, strawberries, carrots, dark leafy greens, and banana, have been known worldwide to contain antioxidants. A characteristic of antioxidants exert both additive and synergistic effects in reducing the risk of chronic diseases. FAV, therefore, exert protective roles against chronic diseases such as cardio and cerebrovascular, ocular and neurological diseases, strokes, cancer, diabetes, hypertension, and blood-related diseases. Several epidemiological studies have presented compelling evidence that the potential of FAV to combat the majority of these health conditions are associated with the natural compounds found in them (Jideani *et al*., 2021).

2.6. Tomato

Tomato (*Solanum lycopersicum L*.) is an important component of the Mediterranean diet. It is a rich source of such micronutrients as carotenoids, vitamin C, folate, and flavonoids that are known to promote human health and wellbeing (Li *et al*., 2016 ; Tommonaro *et al*., 2017 ; Capurso *et al*., 2020 ; Hidalgo-Mora *et al*., 2020).

Carotenoids are a class of compounds containing approximately 600 highly hydrophobic bioactive molecules that are represented in plant taxa. They also serve as pigments, being responsible for the varied and vivid colors present in nature (Campestrini *et al*., 2019 ; Maoka, 2020). Among carotenoids, lycopene (C40H56) is

one of the six essential pigments (β-carotene, α-carotene, γ-carotene, lycopene, zeaxanthin, lutein, and others) discovered starting in 1876, that give the red color to a variety of fruits and vegetables including tomato, watermelon, grapes, apricots, and pink guava (Borel *et al*., 2016 ; Hussain & Sun, 2019). Lycopene is abundant in tomatoes, ranging from 8.8 to 42.0 μg/g fresh weight fruit/vegetable (Sigurdson *et al*., 2017), corresponding up to 90% of the total carotenoid content (Egydio *et al*., 2010 ; Caseiro *et al*., 2020). This pigment seems to protect against chronic degenerative diseases by mitigating oxidative damage (Del Giudice *et al*., 2017).

The lycopene's antioxidant properties are due to its highly unsaturated composition, which consists of an open straight hydrocarbon chain of 11 conjugated and two unconjugated double bonds (Kehili *et al*., 2018). These conjugated double bonds are principally responsible for the characteristic deep red color and for their antioxidant activity (Topal *et al*., 2006). The anti-free radical effect of lycopene in reducing cardiovascular disease was reported to be double that of β-carotene (Müller *et al*., 2016 ; Bacanli *et al*., 2017 ; FG Cicero & Colletti, 2017; Casas *et al*., 2018 ; Saini a *et al*., 2020). Furthermore, carotenoids in general, and lycopene in particular, may act as anti-carcinogenic agents (Mazidi *et al*., 2018 ; Rowles & Erdman, 2020). Indeed, lycopene dietary intake and blood concentration have been negatively associated with cancer, particularly prostate cancer (Wang *et al*., 2016 ; Stice *et al*., 2018 ; Meléndez-Martínez, 2019 ; Bhatt & Patel, 2020 ; Saini b *et al*., 2020). Moreover, epidemiological trials indicate that lycopene also protects against neurodegenerative and such other chronic diseases as asthma, hypertension, osteoporosis, and others (Prema *et al*., 2015 ; Ardawi *et al*., 2016 ; Han & Liu, 2017 ; Bhatt & Patel, 2020 ; Saini a *et al*., 2020). Lycopene is a hydrophobic molecule soluble in fat: in fact, its bioavailability has been estimated at about 23% when pure lycopene was mixed with olive oil (Moran *et al*., 2015), and at about 5% when it was ingested as tomato juice (Cooperstone *et al*., 2015).

The potential availability of antioxidants after the digestion process is critical, and studies reported a partial loss of the bioactive compounds after digestion (Cervantes-Paz *et al*., 2016). Indeed, the bioaccessibility of carotenoids can be affected by many factors, including the food matrix, processing, and cooking methods, and the interactions that occur during digestion and absorption with other dietary compounds, such as fibers, lipids, phytosterols, and other carotenoids (Chacón-Ordóñez *et al*., 2019 ; Yu *et al*., 2019). Among dietary factors, heat and mechanical treatment of foods and

the presence of fat in a meal appear to be critical for carotenoid bioaccessibility and plasmatic bioavailability in vivo (Li *et al*., 2016 ; Salvia-Trujillo *et al*., 2016 ; Bohn, 2018). The carotenoid structure also plays an important role in plasma bioavailability. Lycopene exists in a variety of geometric isomers, including all-trans, mono-cis, and poly-cis forms. The all-trans isomer of lycopene is predominant in fresh tomatoes and difference various tomato-based foods, ranging from 35 to 96% of total lycopene. It is also the most thermodynamically stable form (Schierle *al*., 1997). During tomato processing and storage, lycopene can undergo isomerization. On the other hand, lycopene cis-isomers contribute more than 50% to the total lycopene in human serum and tissue (Arranz *et al*., 2015 ; Khoo *et al*., 2011).

2.7.Tomato powder compounds

Tomato (*Solanum lycopersicon L*) is one of the world's major vegetable with a worldwide production of 126 million tons in 2005 (Kaur *et al*., 2013). Carotenoids are natural yellow, orange, and red pigments found in fruits and vegetables and have a wide range of proposed biologic functions including antioxidant and anticarcinogen properties and immune protection (Campbell *et al*., 2004).

Whereas LYC is the major carotenoid in tomatoes, other carotenoid precursors of LYC, including phytoene (PE) and phytofluene (PF) are also present in substantial amounts (Canene-Adams *et al*., 2005). Carotenoids are a class of lipophilic compounds with a polyisoprenoid structure. Most carotenoids contain a series of conjugated double bonds, which are sensitive to oxidative modification and cis-trans isomerization. There are six major carotenoids (b-carotene, a-carotene, lycopene, b-cryptoxanthin, lutein, and zeaxanthin) that can be found routinely in human plasma and tissues. Among them, b-carotene has been the most extensively studied. More recently, lycopene has attracted considerable attention due to its association with a decreased risk of a number of chronic diseases (Mein *et al*., 2008).

Considerable efforts have been expended in order to identify its biological and physiochemical properties. Relative to b-carotene, lycopene has the same molecular mass and chemical formula, yet lycopene is an open-polyene chain lacking the b-ionone ring structure (Mein *et al*., 2008). Using tomatoes or tomato products, numerous studies have demonstrated decreased DNA damage (Chen *et al*., 2001 ; Bowen *et al*., 2002),

decreased susceptibility to oxidative stress in lymphocytes (Porrini & Riso, 2000), and decreased Low-Density Lipoprotein (LDL) oxidation or lipid peroxidation(Agarwal & Rao, 1998). Porrini & Riso, (2000) refer that the natural balance of carotenoids achieved through normal diets is more effective than carotenoid supplementation. Original and major color materials of tomato are carotenoid, betacarotene and lycopene Some types of carotenoids after consumption can be converted to vitamin A, but lycopene excludes of this property and form approximately 50 % of carotenoids comprise human serum. Otherwise equate diet, the deposits decreases rapidly (Wang *et al*.,1996; Ahuja *et al*., 2006).

2.8. Digestion of Carotenoid

Carotenoid esters are present in considerable amounts in fruits and vegetables but they are not frequently detected in human plasma after ingestion, although the bioavailability of carotenoid esters is considered to be equivalent or even higher than that of free carotenoids. This suggests that hydrolysis of carotenoid esters takes place in vivo, probably in the gastrointestinal lumen and/or within enterocytes, but part of ingested carotenoid esters may also pass intact through the digestive system. These events were not successfully reproduced so far by in vitro digestion protocols, carotenoid esters are detected in the highly variable range in the bioaccessible fraction (Petry & Mercadante, 2019).

Humans ingest no less than 40 carotenoids from common fruit and vegetables. However, the major carotenoids found in human tissues are limited to the following: β-carotene, α-carotene, lycopene, β-cryptoxanthin, lutein and zeaxanthin (Nagao, 2014).

2.8.1.Solubilization

At an early stage of digestion, carotenoids must be released from food matrices. The carotenoids released must be effectively dispersed in the digestive tract, and finally, solubilized in mixedmicelles (Nagao, 2014). Carotenoids released from food matrices are dispersed with the aid of bile, which contains bile salts and phosphatidylcholine. Thereafter, carotenoids are solubilized in mixed-micelles, which are formed through hydrolysis of lipids emulsified in digesta by lipolytic enzymes in pancreatic juice. The mixed-micelles comprise bile acids, cholesterol, lysophosphatidyl choline, fatty acid and monoacylglycerol. A portion of the dietary carotenoids solubilized in the mixed-

micelles is taken up into the intestinal epithelia, meaning solubilization of carotenoids in the mixed-micelles is an important process for bioavailability (Nagao, 2014).

Intestinal absorption carotenoids solubilized in mixed-micelles are taken up by the epithelial cells of the jejunum and incorporated into the chylomicron, and with chylomicron secreted into the lymph and circulated within the body (Yonekura & Nagao 2007) (Fig. 2- 2). The transfer of carotenoids from mixed-micelles to intestinal cells was thought to be mediated by simple diffusion dependent on the concentration gradient across the cellular membrane. When carotenoids solubilized in the mixed-micelles compatible with those formed in the intestine were incubated with human intestinal Caco-2 cells, a positive relationship was found between the hydrophobicity of carotenoids and the amount of carotenoid taken up by the cells (Sugawara *et al*., 2001).

2.8.2.Metabolism

In general, animals do not synthesize carotenoids, and so those found in animals are either directly obtained from food or partly modified through metabolic reactions. The major metabolic conversions of carotenoids found in these animals are oxidation, reduction, translation of double bonds, oxidative cleavage of double bonds, and cleavage of epoxy bonds. It is well-known that carotenoids that contain unsubstituted β-ionone rings such as β-carotene, α-carotene, β-cryptoxanthin. and β,ψ-carotene (γ-carotene) are precursor of retinoids and are called pro-vitamin A. Furthermore, carotenoids in animals play important roles such as photo-protectors, antioxidants, enhancers of immunity, and contributors to reproduction. Several animals use carotenoids as signals for intra-species (sexual signaling, social status signaling, and parent–offspring signaling) and inter-species (species recognition, warning coloration, mimicry, and crypsis) communication (Maoka, 2020).

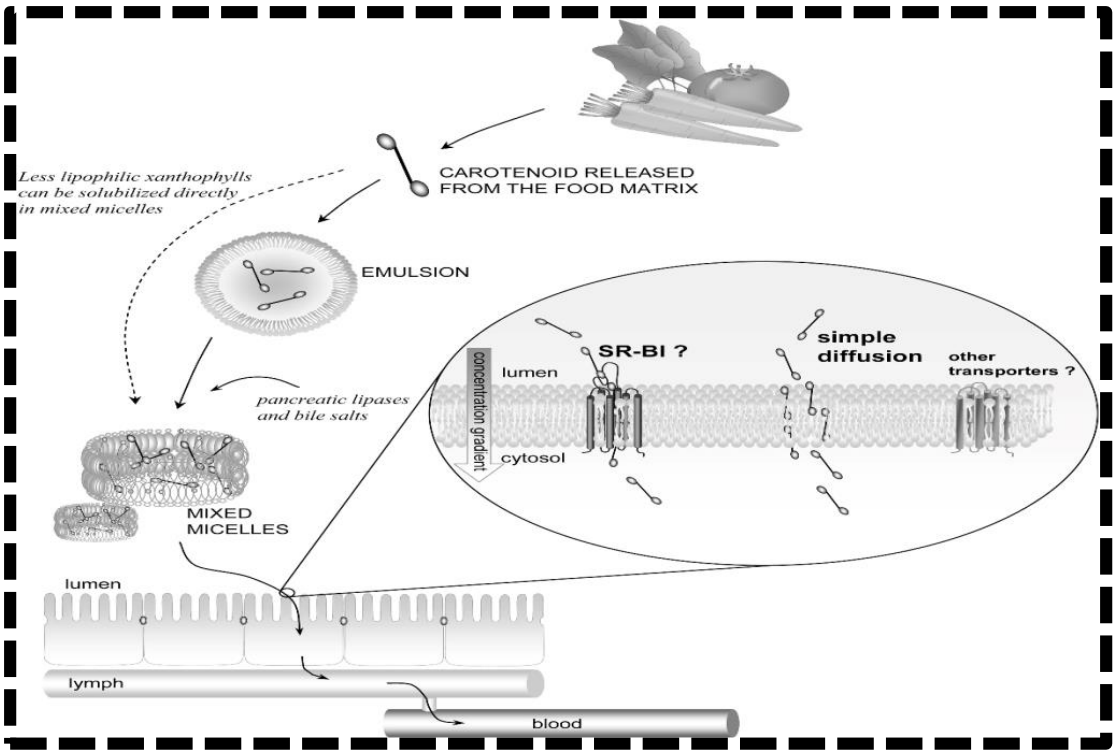

Fig. (2-2) Scheme of dietary carotenoid absorption (Yonekura & Nagao, 2007)

2.9. Lycopene

Lycopene (LYC) is a natural food-derived pigment belonging to carotenoids used in food processing, and is mainly enriched in FAV with a red color, such as tomatoes, watermelon, and papaya (Liang *et al*., 2019). Lycopene is a major dietary carotenoid protecting cells against oxidative damage to lipids, proteins, and DNA (Cheng *et al*., 2019 ; Han *et al*., 2019).

The LYC has been identified as a class A nutrient by the World Health Organization. The LYC can be used as a dietary botanical bioactive substance with various bioactivities, including antioxidant capacity, and has therapeutic potential against diseases (Grabowska *et al*., 2019 ; Liang *et al*., 2019). The LYC or LYC-rich materials have been reported to exhibit mitochondrial protective effects against toxicity damage caused by hydrogen peroxide (H2O2) (Reshmitha *et al*., 2017),

D-galactosamine/lipopolysaccharide (D-GalN/ LPS) (Sheriff *et al*., 2017), and 1-methyl-4-phenylpyridinium ion (Yi *et al*., 2013).

2.10. Antioxidants

Antioxidants is a molecules that inhibits the oxidation of other molecules. Oxidation is a chemical reaction that can produce free radicals. Antioxidants can be categorized in to enzymatic and nonenzymatic (Agarwal & parbakaran, 2005). The enzymatic antioxidant includes catalase (CAT), superoxide dismutase (SOD), glutathione reductase (GSH-r) and glutathione peroxidase (Gpx). The enzymatic antioxidant act as scavengers for free radicals from both intracellular and extracellular origin and there for preventing lipid peroxidation of plasma membrane, whereas the nonenzymatic antioxidant Carotenoids, together with vitamins C and E and phenolic compound are the major antioxidants of plant-derived foods. can be used to moderate the negative side effects of environmental origin stress (Nojoku, 1986 ; Garg & Bansal, 2000).

A number of studies have shown that the supplementation of nutrient antioxidants like carotenoids may enhance the stability and extend the shelf life of foods. This antioxidant activity is also the most cited mode of action of carotenoids in the prevention of chronic degenerative diseases (Rodriguez-Amaya, 2015).

2.10.1. Superoxide dismutase (SOD)

Superoxide dismutase (SOD) provide an important role as cellular defense enzyme against free radical damage (Pillai and Pillai, 2002). SOD extracellular as antioxidant enzyme can catalyze the dismutation of the superoxide anion (the high reactive species) to O2 and to H2O2 (the less reactive species). Then H2O2 can be destroyed by action of CAT or GPX reactions (Costa *et al*., 2009).

O2- + O2- +2H- + SOD > H2O2 +O2

Three forms of SOD has been found: mitochondrial Mn-SOD, cytosolic Cu/Zn-SOD, and extracellular SOD (Lindberg *et al*., 2005 ; Ding & Dokholyan, 2008) Mn-SOD is the biological importance one. Cu/Zn-SOD is believed to play an important role as the first antioxidant defense line (Ding & Dokholyan, 2008).

2.10.2. Malondialdehyde (MDA)

It is the organic compound with the nominal formula CH2(CHO)2. A colorless liquid, malondialdehyde is a highly reactive compound that occurs as the enol, it occurs naturally and is a marker for oxidative stress. Malondialdehyde mainly exists as the enol.

CH2(CHO)2 → HOC(H)=CH-CHO (Nair *et al*., 2008).

2.10.3.Glutathione peroxidase (Gpx)

It is the general name of an enzyme family with peroxidase activity whose main biological role is to protect the organism from oxidative damage, The biochemical function of glutathione peroxidase is to reduce lipid hydroperoxides to their corresponding alcohols and to reduce free hydrogen peroxide to water (Muthukumar *et al*., 2011). Glutathione peroxidase was discovered in 1957 by Gordon C. Mills (Mills, 1957).

Chapter Three: Methodology

3. Materials and Methods

3.1. Birds and management

One day old broiler chicks (Ross 308) were purchased from a local hatchery in Karbala (Barakat of Imam Hussein), the chicks were raised according to routine management practice. Basal diet and water were supplied *ad libitum* to meet National Research Council NRC, (1994).

3.2. Diet preparation

Basal diet obtained from local factory (sahul al-khaerate) for supplied poultry diet in Karbala The composition and chemical analysis of the basal diet is presented in Table (3-1), the feed contained AF 30 PPb obtained from bad storage (Kana *et al*., 2014) **In appendix 1.**

Table (3-1). Ingredients and of chemical analysis experimental diets.

Ingredient % Starter (1-21 day))	Starter (1-21 day)	Finisher (22-35 day)
Corn	29	30
Soya bean meal(44% protein)%	28	20
Wheat%	27.5	35.5
Animal Protean (50%)	10	10
Oil%	3	3
Salt%	0.3	0.3
Tomato powder%	1	0
Limestone%	1.2	1.2
Total	100	100

3.3. Experimental Design

One day old broiler chicks (Ross 308) were used in the present study birds were randomly divided into 5 equal groups of 20 birds for each group as following: figure (3-1).

***The first group: (Group 1)** birds in this group were fed from first day to end of experimental on basal diet without any additive as a control group.

***The second group: (Group 2)** birds of this group were fed from first day to end to experimental on contaminated diet AF 30 ppb only without any treated.

***The third group: (Group 3)** birds of this group were fed from first day to end to experimental on contaminated diet AF 30 ppb supplemented with 1 kg/ton Mycofix.

***The fourth group: (Group 4)** birds of this group were fed from first day to end of experimental on contaminated diet AF 30 ppb supplemented with tomato powder 10 kg/ton TP.

***The fifth group: (Group 5)** birds in this group were fed from first day to end of experimental on basal diet with addition tomato powder 10 g/kg TP .

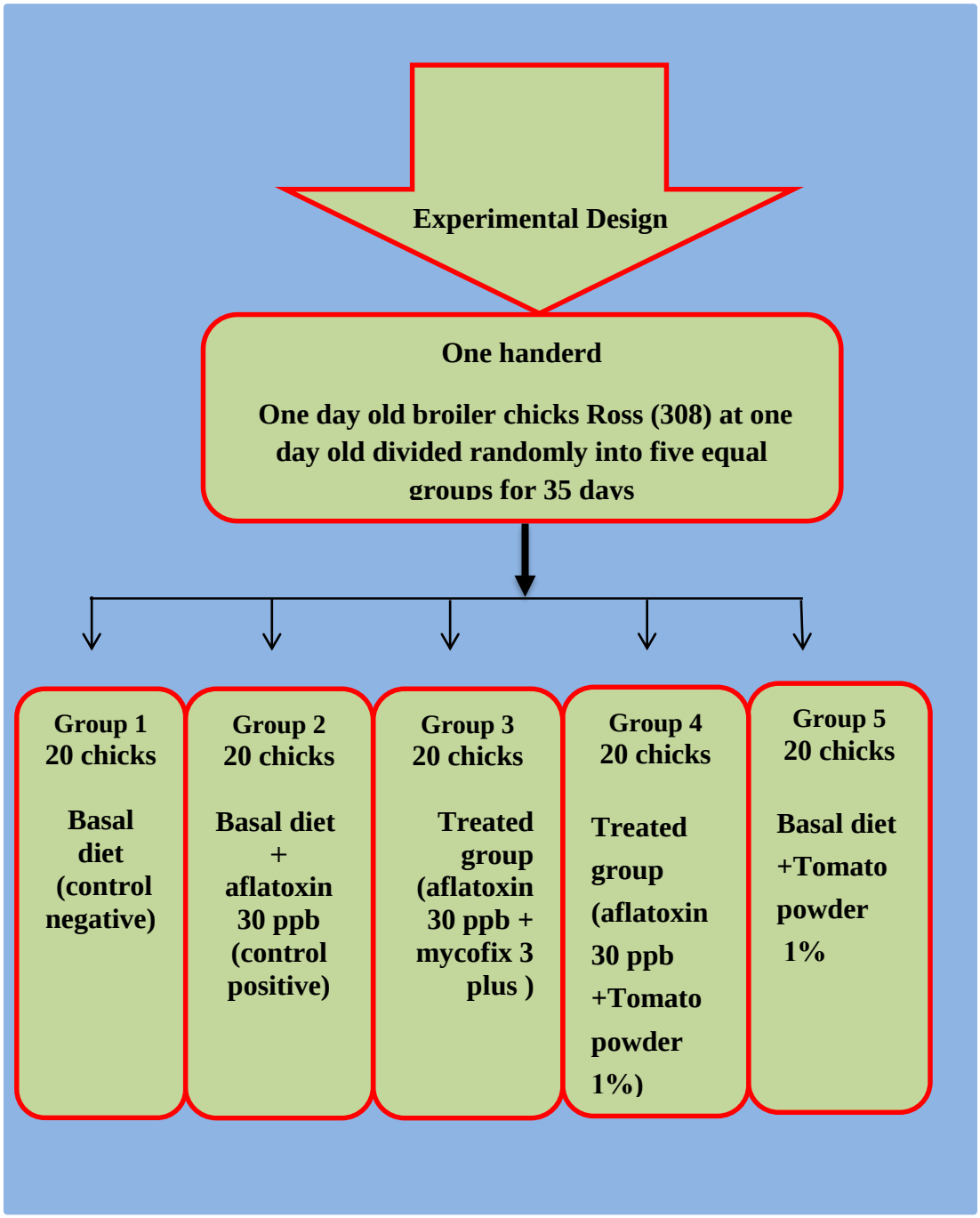

Figure (3-1) Experimental design

3.4. Vaccination Programe

All live attenuated vaccines (1000 dose) were prepared with distal water, and were given by comparing the number of birds multiplied by the bird's age, in which given by the water.

Number of birds × bird's age = amount of water (ml)

1) Live attenuated Newcastle Disease (ND) virus vaccine LaSota strain. The vial contains (1000 dose) (Boehringer - Germany) were administered by (Drinking water).

2) Live attenuated Infectious Bursal Disease (IBD) virus Lstrain. The vial contains (1000 dose) (Ceva – Hungary) were administered by (Drinking water) (Table 3-2).

Table (3-2) vaccines, which used in this study

Age (days)	Strain	Origin	Route of administration
(10 & 20 days)	ND La sota	Boehringer Germany	Drinking water
(14 days)	IBD GumboL	Ceva -Hungary	Drinking water

3.5. Preparation of tomato powder

Fresh, mature, and ripe tomatoes (*Lycopersicon esculentum*) were purchased from local market(approximately 50 KG). The tomatoes were cut into slices (Arslan & Özcan, 2011). Tomato slices were distributed uniformly as a thin layer onto the stainless steel trays and dried under direct sunlight (Balladin & Headley, 1999).

3.5.1. Calculation of concentration of Tomato sample

$$\text{Concentration of sample mg/ml} = \frac{\text{Area of sample}}{\text{Area of standard}} \times \text{Conc. of standard} \times \text{dilution factor}$$

$$\text{Concentration \%} = \frac{\text{Wight of part}}{\text{Wight of all}} \times 100$$

3.6. Analysis of AF residue in liver and breast muscles by HPLC

The samples (25g) were sonicated in 100 mL 70:30 v/v methanol : water for 40 min and centrifuged for 5 min, then 5 mL of the supernatant was drawn, diluted with 20 mL water, and passed through the immune affinity column at no more than 3 mL/min (the column was pre-viously conditioned with 20 mL distilled water). The column was rinsed with 10 mL distilled water to remove the matrix components and the dried by passing air through to remove any remaining water. The final quantitative elution was accomplished by adding methanol (1.4 mL) on to the column and flushing with air. The eluate was diluted to 2 mL with water and passed through a 0.45 mm filter, and the filtrate was injected into the HPLC (Liu *et al., 2012).*

3.7. Production parameters

3.7.1. Weekly body weights

The chicks were weighed individually on days 1st, 7th, 14th, 21th, 28th and 35th. The live body weights for each treatment were recorded (Al-Zubaidi, 1986).

3.7.2. Weekly weight gains

The weekly body weight gain were calculated depending on the following equation:-

Weekly weight gain (gm) = body weight at the end of the week –body weight at the beginning of the week (Al-Zubaidi, 1986).

3.7.3. Weekly feed consumption

Feed intake was measured weekly depending on equation mentioned by (Al-zubaidi, 1986) and that was done by weighing remained feed at the end of each week and subtracted it from the total quantity offered at the beginning of the week.

3.7.4. Weekly feed conversion ratio

Weekly feed conversion ratio was measured for each group in the experiment as the equation mentioned by (Al-Zubaidi, 1986).

$$\text{Weekly feed conversion} = \frac{\text{average weekly feed intake (gm)}}{\text{average weekly body weight gain (gm)}}$$

3.8. Collection of blood samples

At day 4, 28 and 35th of age, blood samples were collected from 5 birds in each group from the jugular vein (2 ml) in a test tube without anticoagulant (plane tube) the serum was separated by centrifugation for 10 minutes at 3000 rpm and stored in a freeze (-20) until analysis to determine the biochemical and immunological tests.

3.9. Weight of the liver, spleen, thymus and bursa of fabricius tissues samples

On day 28 and 35, five birds from each group were weighed. The birds were sacrificed by cervical dislocation and collect its certain tissues for weighting.

3.10. Hepatosomatic index

The body weight (gm) and liver weight (gm) were recorded for each individual bird in 35th day, and their indices were calculated according to (Sellers *et al.*, 2007 ; Williams & zedek, 2010) as follow:

Organ index = organ weight (g) / body weight (g) × 100

3.11. Sensory analysis

Sensory evaluation was done by taking meat slices from breast with a size of 2 cm 3 and cooked in an electric oven at a temperature of 177 ° C then lowered to the sensory evaluation. In the sensory evaluation share five adults women, the sensory evaluation was done for flavor, Tenderness, juicy, color and palatability according to the scores shown in the table (3-3) (Al–Fayadh *et al.*, 2005).

Table (3-3) Sensory evaluation

appreciation	Tenderness	Juicy	Color	Flavor	Palatability
1	not tenderness	very dry	very light brown	very unpleasant flavor	very unpalatable
2	less tenderness	dry	light brown	unpleasant flavor	unpalatable
3	average tenderness	average	medium brown	Medium flavor	average palatability
4	tenderness	juicy	dark brown	desirable flavor	palatable
5	very tenderness	very juicy	very dark brown	Very desirable flavor	Very palatable

3.12. Determination of antibody titter Newcastle Disease (ND) virus and Infectious Bursal Disease (IBD) virus by ELISA test

The procedure used in this test was performed according to the manufacturer instructions listed in the ProFLOK® (ND and IBD) ELISA Kit (Synbiotics–USA) (Lamichhane *et al.*, 1997) **in appendix 2.**

3.13. Biochemical parameter

3.13.1 Enzymes concentration

Serum Aspartate aminotransferase activity (AST) and Serum Alanine aminotransferase activity (ALT) is determined by using a kit (SPECTRUM AST and ALT – kit, Egypt) (Young, 1990) **in appendix 3**.

3.13.2. Serum Creatinine concentration

Creatinine reacts with picric acid under alkaline condition to form a yellow-red complex (Tietz, 1986) **in appendix 4**.

3.13.3. Antioxidant status

3.13.3.1. Measurement of serum Malondialdehyde concentration (MDA)

The level of malondialdehyde was determined by a modified procedure described (Guidet & Shah, 1989) **in appendix 5.**

3.13.3.2. Measurement of serum Glutathione Peroxidase concentration (Gpx)

Measurement of serum Gpx was done by using ELISA kit (Comhair *et al.*, 1999) **in appendix 5.**

3.13.3.3. Measurement of serum Superoxide dismutase concentration (SOD)

The procedure was done according to the instructions of the manufacture of ELISA Kit -Elabscience biotechnology/ china **in appendix 5.**

3.13.4. Measurement of lipid profile tests

3.13.4.1. Estimation of serum total Cholesterol concentration (mg/dL)

Cholesterol concentration was measured by using Cormay cholesterol kit. oxidation and after enzymatic hydrolysis, the cholesterol is determined in the presence of phenol and peroxidase,4-aminoantipyrine and the hydrogen peroxide forming quinoneimine the indicator (Fasce, 1982) **in appendix 6.**

3.13.4.2. Estimation of serum Triglyceride concentration (mg/dL)

Triglyceride concentration was measured by Cormay triglyceride kit. Its hydrolyzed to glycerol enzymatically according to the following reaction (Fossati & Prencipe, 1982) **in appendix 6.**

3.13.4.3. Estimation of serum HDL-Cholesterol concentration (mg/dL)

HDL-Cholesterol concentration was measured by using Cormay HDL kit. The supernatant contains high density lipoprotein (HDL). The HDL-cholesterol is then

spectrophotometrically measured by means of the coupled reaction described by (Grove, 1979) **in appendix 6.**

3.13.4.4. Estimation of serum LDL-Cholesterol concentration (mg/dL)

LDL-C was measured by using Cormay LDL kit (Alan, 2006) **in appendix 6.**

3.13.4.5. Estimation of serum VLDL-Cholesterol concentration (mg/dL)

VLDL-C was measured by using the following equation **VLDL = TG /5**

(Friedewald, 1972).

3.14. Histopathological Technique

The small part of liver of each animal were quickly removed for pathological study. The samples from each section were immersed in 10% formaldehyde solution then prepared for pathological study according to Mescher method (2013) with aid of the light microscope.

3.15. Instruments: The instruments used in this study are summarized in the table (3-4)

Table (3-4) instruments that used in study

NO.	Equipment & Instrument	Company	Country
1.	Electronic balance	Sf-400	India
2.	Centrifuge	Hettich Roto fix11	Japan
3.	Digital camera	Toup Cam	China
4.	HPLC	Sykam	Germany
5.	ELISA reader	Bio Kit	USA
6.	ELISA washer	Bio Kit	USA
7.	Spectrophotometer	Labomed	UK
8.	Micropipette 100-1000 µl	Cyan	Germany

9.	Light microscope	Leica	China
10.	Manual Plastic drinkers(3Liters)	Local Market	Iraq
11.	Manual Plastic Feeders(3Kg.)	Local Market	Iraq
12.	Freezer	Newal	Turkish
13.	Sterile syringes 3,5ml	Proton	Malaysia

3.16. Chemicals

The chemicals and its origin are illustrated in this table

Table (3-5) chemicals materials and solutions used

No.	**Chemicals**	**company**	**Country**
1.	ALT– kit	Spectrum	Egypt
2.	AST– kit	Spectrum	Egypt
3.	Creatinine estimation kit	Spectrum	Egypt
4.	Gpx	Abcam	USA
5.	MDA	Biotechnology	China
6.	SOD	Biotechnology	China
7.	Buffered formalin 10%	BDH	England
8.	Ethanol absolute (99%)	Barcelona	Spain

9.	Paraffin Wax.	Local Market	Iraq
10.	Ethyl alcohol absolute (90%)	Local Market	Iraq
11.	Blood lipid profiles kit	Cormay Kit	Poland
12.	Hematoxylin and Eosin stain (H&E)	BDH	England
13.	ND and IBD Kit	Synbiotics	USA
14.	Xylne absolute	BDH	England
15.	Tomato	Local Market	Iraq

3.17. Statistical Analyses

The data were analyzed with SPSS (16.0 for Windows) by using a one-way analysis of variance (ANOVA). Differences between means were determined using Tukey's test in which the significance level was designated at ($P < 0.05$).

Chapter Four: Results and Analysis

4. Results and analysis

4.1.Analysis of tomato powder contents in HPLC

2 gm of tomato powder were macerated in 20 mL of 50 % methyl alcohol (MeOH), The mixtures were sonicated in an ultrasonic bath for 10 minutes . The extracts were filtered through filter paper, evaporated (to 1 mL) by using stream of liquid nitrogen and filtered through a 0.45 mm cellulose filter (Millipore). The filtrate was then transferred into a vial and filled up with 50 % MeOH to a volume of 1.0 mL. 20 ul were injected on HPLC column and the concentration were calculated by comparing the peak area of standard with that of sample at the same separation conditions (Pataro *et al.*, 2018), the results in table (4-1).

Table (4-1) HPLC analysis of tomato powder compounds

No.	Subjects	Retention time minute	Area µv	Concentration %
1	Protein	4.58	3562.015	1.6
2	Lipid	1.970	123.835	1.7
3	CHO	2.563	674.035	2.5
4	Fiber	7.477	4286.621	32.8
5	Lycopene (ppm)	7.253	413.565	90.56
6	Vit C (ppm)	4.464	127.034	122.6
7	Vit E (ppm)	6.193	581.522	55.9

4.2. Productive performance of broilers

4.2.1. Effect of TP on the body weight (gm) of broilers

A significant decrease ($P<0.05$) was noticed in the body weight in the AF group in the ages of 7,14,21 and 28 days in comparison with the other treated groups (table 4-2-1). In addition, had no statistically significant ($P>0.05$) among all groups in age 1and 35 day.

Table (4-2-1) Effect of TP on the body weight (gm) of broilers (Mean ± SD)

Ages Groups	Day 1	Day 7	Day 14	Day 21	Day 28	Day 35
Group 1 (CON)	50.1± 2.4 A	173.0± 15.7 A	538.5± 51.7A	1140.3± 93.6A	1643.1± 139.0A	2093.7± 366.8 A
Group 2 (AF)	52.2± 4.1A	152.5± 24.7B	487.0± 59.8B	917.7± 100.9B	1435.4± 173.0B	1907.7± 130.5A
Group 3 (AF+M)	52.8± 3.6A	166.8± 7.2AB	517.0± 35.4AB	979.5± 110.7B	1506.3± 114.5AB	1982.1± 187.4A
Group 4 (AF+TP)	47.7± 3.2A	170.9± 18.8A	532.2± 47.0A	1095.2± 95.4A	1593.0± 190.3A	2053.8± 194.8A
Group 5 (TP)	49.2± 3.6A	177.4± 20.0A	551.6± 43.7A	1165.0± 45.9A	1657.7± 132.1A	2203.8± 151.6A

Difference letters in the same column represent a significant difference at (P<0.05)

4.2.2. Effect of TP on the feed intake (gm) of broilers

Table (4-2-2) Effect of TP on the feed intake (gm) of broilers

Ages Groups	Day 7	Day 14	Day 21	Day 28	Day 35
Group 1 (CON)	113	345	581.5	1057.667	1413.333
Group 2 (AF)	143	347	706.5	1031.333	1473.333
Group 3 (AF+M)	109	383	710	1242.333	1588.667
Group 4 (AF+TP)	146	421	729.5	1250	1600
Group 5 (TP)	138	358	658.25	1144.667	1486.667

4.2.3. Effect of TP on the body weight gain (gm) of broilers

Table (4-2-3) statistically showed that there were a significant increases (P<0.05) in body weight gain of broilers in the group AF&TP in Age 28 days and in the group TP in Ages 7,21 days in comparison with the AF group, While AF group showed decrease significantly (P<0.05) in body weight gain of broilers in ages 7,21 and 28 days

compared with other treated groups. Also it noticed in the same table no significant difference at (P>0.05) in ages 14 and 35.

Table (4-2-3) Effect of TP on the body weight gain (gm) of broilers (Mean ± SD)

Ages Groups	Day 7	Day 14	Day 21	Day 28	Day 35
Group 1 (CON)	120.0616.6 AB	365.5±50.5 A	601.8±96.9 A	502.8±60.2 AB	450.6±108.2 A
Group 2 (AF)	101.0 ±29.6 B	332.0±59.6 A	478.1±62.9 B	429.2±77.7 B	605.6±157.6 A
Group 3 (AF+M)	114.2± 9.1 AB	347.0±32.8 A	434.3±97.7 B	557.7±85.9 AB	461.6±100.0 A
Group 4 (AF+TP)	118.5±19.8 AB	350.3±70.6 A	528.0±83.8 AB	619.5±100.0 A	537.6±129.0 A
Group 5 (TP)	129.0± 20.7 A	377.6±45.0 A	605.9±66.2 A	497.5±94.7 AB	543.3±109.3 A

Difference letters in the same column represent a significant difference at (P<0.05)

4.2.4. Effect of TP on the feed conversion rate (FCR) of broilers

Table (4-2-4) Effect of TP on the feed conversion rate (FCR) of broilers

Ages Groups	Day 7	Day 14	Day 21	Day 28	Day 35
Group 1 (CON)	1.17	1.17	1.28	2.10	3.13
Group 2 (AF)	2.39	1.74	2.39	3.08	3.09
Group 3 (AF+M)	1.36	1.57	2.33	2.38	3.68
Group 4 (AF+TP)	1.63	1.60	2.04	2.01	2.97
Group 5 (TP)	1.42	1.26	1.44	2.30	2.73

4.3. Weight of liver, spleen, thymus and bursa of fabricius of broilers

4.3.1. Effect of TP on weight of liver, spleen, thymus and Bursa of fabricius of broilers at 28 day

A significant increase (P<0.05) was noticed in the weight of liver in the AF group respectively compared with the other treated groups, in the same table the results showed a significant decrease (P<0.05) in the weights of the spleen, thymus and bursa of fabricius in the AF group compared with other treated groups shown in (table 4-3-1).

Table (4-3-1) Effect of TP on weight of liver, spleen, thymus and bursa of fabricius of broilers at 28 day (Mean ± SD)

Difference letters in the same column represent a significant difference at (P<0.05)

Parameters Groups	Liver (g)	Spleen (g)	Thymus (g)	Bursa (g)
Group 1 (CON)	14.1 ± 0.2 B	1.3 ± 0.2 A	3.3 ± 0.7 A	1.6 ± 0.1 A
Group 2 (AF)	19.1 ± 0.8 A	0.6 ± 0.1 B	1.7 ± 0.2 B	0.6 ± 0.2 B
Group 3 (AF+M)	15.1 ± 0.4 B	1.1 ± 0.2 A	3.1 ± 0.8 A	1.1 ± 0.3 AB
Group 4 (AF+TP)	14.1 ± 0.7 B	1.2 ± 0.1 A	3.3 ± 0.4 A	1.3 ± 0.4 A
Group 5 (TP)	14.0 ± 0.8 B	1.3 ± 0.3 A	3.2 ± 0.7 A	1.4 ± 0.1 A

4.3.2. Effect of TP on weight of liver, spleen, thymus and bursa of fabricius of broilers at 35 day

A significant increase (P<0.05) was noticed in the weight of liver in the AF group compared with the other treated groups (table 4-3-2). It also noticed in the same table no significant difference at (P>0.05) in the weights of spleen, thymus and bursa of fabricius among all groups.

Table (4-3-2) Effect of TP on weight of liver, spleen, thymus and bursa of fabricius of broilers at 35 day (Mean ± SD)

Parameters Groups	Liver (g)	Spleen (g)	Thymus (g)	Bursa (g)
Group 1 (CON)	27.6 ± 1.1 B	1.4 ± 0.1 A	3.3 ± 0.5 A	1.0 ± 0.3 A
Group 2 (AF)	31.6 ± 1.0 A	1.3 ± 0.2 A	3.8 ± 0.6 A	0.8 ± 0.1 A
Group 3 (AF+M)	29.3 ±1.4 B	1.5 ± 0.2 A	3.5 ± 0.7 A	1.1 ± 0.5 A
Group 4 (AF+TP)	29.0 ± 1.3 B	1.70 ± 0.4A	4.1 ± 1.6 A	1.2 ± 0.4 A
Group 5 (TP)	28.5 ± 1.1 B	1.6 ± 0.5 A	4.0 ± 1.0 A	1.1 ± 0.3 A

Difference letters in the same column represent a significant difference at (P<0.05)

4.3.3. Effect of TP on the Hepatosomatic Index of broilers at 28 and 35 day

A significant increase (P<0.05) was noticed in liver index in the AF group compared with other treated groups.

Table (4-3-3) Effect of TP on the Hepatosomatic Index of broilers at 28 and 35 day (Mean ± SD)

Index Group	Liver Index % Day 28	Liver Index % Day 35
Group 1 (CON)	0.80 ± 0.03 C	1.44 ± 0.12 B
Group 2 (AF)	1.36 ± 0.11 A	1.64 ±0.11 A
Group 3 (AF+M)	1.00 ± 0.03 B	1.42 ± 0.06 B
Group 4 (AF+TP)	0.84 ± 0.05 C	1.41 ± 0.03 B
Group 5 (TP)	0.80 ± 0.05 C	1.38 ± 0.08 B

Difference letters in the same column represent a significant difference at (P<0.05)

4.4. Sensory traits of broilers

A significant increase (P<0.05) was noticed in the sensory traits of tenderness, juicy, flavor, color and palatability in the AF&TP, AF&M and TP groups respectively compared with the AF group shown in (table 4-4).

Table (4-4) Effect of TP on some Sensory traits of broilers at 35 day (Mean ± SD)

Parameters Group	Tenderness	Juicy	Color	Flavor	Palatability
Group 1 (CON)	4.4 ± 0.5 A	3.4 ±0.8 A	3.2±0.8A	4.8 ± 0.4A	4.8± 0.4A
Group 2 (AF)	2.0 ± 0.7 B	1.6 ± 0.5 B	1.0 ±0.0B	2.4 ±0.5C	2.6 ± 0.5B
Group 3 (AF+M)	3.4 ±0.5 A	2.6 ± 0.4AB	3.6 ±1.3A	.36± 0.5B	.38 0.4A
Group 4 (AF+TP)	3.6 ± 0.8A	3.2 ±0.4 A	3.0±0.7 A	4.2 ± 0.4AB	4.0 ± 0.7A
Group 5 (TP)	3.8 ± 0.8 A	.34 ± 0.7 A	2.6±0.5 A	4.0 ±0.7AB	4.0 ± 0.8A

Difference letters in the same column represent a significant difference at (P<0.05)

4.5. Immunologically status of broilers

4.5.1. Effect of TP on Newcastle disease (ND) antibody titer of broilers

Table (4-5-1) showed that their were significant increase (P<0.05) in the AF&TP group in ND titer of broilers at age 28 and 35 days with significant decrease (P<0.05) in the AF group in ND titer of broilers at age 28 and 35 days compare with the other treated groups. Also it noticed in the same table no significant difference in ND titer between all groups at age 4 days of chicks.

Table (4-5-1) the effect of TP on Newcastle disease (ND) antibody titer of broilers (Mean ± SD)

Ages Groups	ND at 4 days	ND at 28 days	ND at 35 days
Group 1 (CON)	9301.0 ± 386.0 A	3842.6 ± 331.6 A	4203.6 ± 306.6 A
Group 2 (AF)	9160.0 ± 454.6 A	1702.2 ± 323.6 B	2006.0 ± 87.5 B
Group 3 (AF+M)	9310.0 ± 487.8 A	3369.2 ± 281.2 A	4018.2 ± 498.7 A
Group 4 (AF+TP)	9134.4 ± 443.6 A	3409.0 ± 378.3 A	4214.0 ± 477.9 A
Group 5 (TP)	9290.0 ± 410.5 A	3788.4 ± 395.4 A	4109.0 ± 202.9 A

Difference letters in the same column represent a significant difference at ($P<0.05$)

4.5.2. Effect of TP on Infectious Bursal disease (IBD) antibody titer of broilers

Table (4-5-2) showed that their were significant increase ($P<0.05$) in the AF&TP group in IBD titer of broilers at age 28 and 35 days with significant decrease ($P<0.05$) in the AF group in IBD titer of broilers at age 28 and 35 days compare with the other treated groups. Also it noticed in the same table no significant difference in IBD titer between all groups at age 4 days of chicks.

Table (4-5-2) the effect of TP on Infectious Bursal disease (IBD) antibody titer of broilers (Mean ± SD)

Ages Groups	IBD at 4 days	IBD at 28 days	IBD at 35 days

Group 1 (CON)	14986.8 ±256.9 A	11724.2 ±581.1 A	14940.2 ± 642.1 A
Group 2 (AF)	14954.2 ± 93.7 A	4431.6 ± 407.6 B	11821.0± 912.7 B
Group 3 (AF+M)	14886.4 ±339.8 A	10978.4 ±673.1 A	14319.20±1188.2A
Group 4 (AF+TP)	15367.6 ±459.5 A	11146.4 ±368.3 A	14522.2 ±1219.2 A
Group 5 (TP)	14983.6 ±221.7 A	11865.6 ±202.7 A	16036.2 ± 548.3 A

Difference letters in the same column represent a significant difference at (P<0.05)

4.6. Biochemical parameters of broilers

4.6.1. Effect of TP on AST, ALT and Creatinine of broilers at 28 day

A significant decrease (P<0.05) was noticed in the concentrations of AST, ALT and creatinine enzymes in the AF&TP group compared with the AF group, while in the AF group had a significant increased (P<0.05) in the concentration of enzymes compared with other treated groups show in table (4-6-1).

Table (4-6-1) Effect of TP on AST, ALT and Creatinine of broilers at 28 day (Mean ± SD)

Parameters Groups	AST U/L	ALT U/L	Creatinine mg/dl
Group 1(CON)	160.2 ± 5.1 D	4.4 ± 1.1 B	0.3 ± 0.03 C
Group 2 (AF)	356.6 ± 36.8 A	44.4 ± 5.3A	0.7 ± 0.07 A
Group 3 (AF+M)	278.8 ± 20.0 B	5.0 ± 1.5 B	0.4 ± 0.05 B
Group 4 (AF+TP)	220.0 ± 17.3 C	5.5 ± 1.2 B	0.4 ± 0.03 B
Group 5 (TP)	172.8 ± 17.5 D	5.4 ± 1.1 B	0.3 ± 0.01 C

Difference letters in the same column represent a significant difference at (P<0.05)

4.6.2. Effect of TP on AST, ALT and Creatinine of broilers at 35 day

A significant decrease (P<0.05) was noticed in the concentrations of creatinine in the AF&TP group compared with the AF group, while in the AF group had a significant increase (P<0.05) in the concentration of enzymes compared with other treated groups table (4-6-2). Also it was noticed that there is no significant difference at (P>0.05) in the enzyme ALT among all groups.

Table (4-6-2) Effect of TP on AST, ALT and Creatinine of broilers at 35 day (Mean ± SD)

Parameters Groups	AST U/L	ALT U/L	Creatinine mg/dl
Group 1(CON)	164.2 ± 2.9 B	7.4±0.8A	0.3±0.02C
Group 2 (AF)	188.0 ± 6.5 A	8.2 ± 0.8A	0.6 ± 0.02A
Group 3 (AF+M)	183.8 ± 16.8AB	7.4 ± 2.7A	0.38± 0.05BC
Group 4 (AF+TP)	172.4 ± 13.1AB	6.6 ± 1.1A	0.3 ± 0.01C
Group 5 (TP)	166.8 ± 11.1AB	7.0 ± 1.4A	0.33± 0.02C

Difference letters in the same column represent a significant difference at (P<0.05)

4.6.3. Effect of TP on Blood lipid profiles of broilers at 28 day

A significant decrease (P<0.05) was noticed in the serum concentration of cholesterol, triglyceride, LDL and VLDL at the AF&TP, AF&M and TP groups respectively compared with the AF group (table 4-6-3), while HDL was increased significantly (P<0.05) in the same groups.

Table (4-6-3) Effect of TP on Blood lipid profiles of broilers at 28 day (Mean ± SD)

Parameters Groups	Cholesterol mg/dl	Triglyceride mg/dl	HDL mg/dl	VLDL mg/dl	LDL mg/dl

Group1 (CON)	115.2 ±3.8 C	56.6 ± 2.0 C	55.4±1.1 A	11.5 ±0.4 C	50.2 ±3.1 C
Group 2 (AF)	146.2 ± 3.1 A	83.4 ± 4.9 A	42.4 ±2.0 B	16.6±0.9 A	87.1 ± 5.5A
Group 3 (AF+M)	128.2 ± 2.5 B	66.8 ± 3.7 B	51.2±4.6 A	13.3 ±0.7 B	63.6 ±5.0 B
Group 4 (AF+TP)	119.6 ± 3.3C	61.4 ±4.6 BC	52.2 ±2.5 A	12.2 ±0.9 C	55.1 ±2.8 C
Group 5 (TP)	118.4 ± 3.9 C	57.8 ± 3.8 C	53.0±1.8 A	11.5 ± 1.1 C	53.8 ±4.7C

Difference letters in the same column represent a significant difference at (P<0.05)

4.6.4. Effect of TP on Blood lipid profiles of broilers at 35 day

A significant (P<0.05) decreased was noticed in the serum concentration of cholesterol, triglyceride, LDL and VLDL at the AF&TP, AF&M and TP groups respectively compared with the AF group (table 4-6-4), while HDL was increased significantly (P<0.05) in the same groups.

Table (4-6-4) Effect of TP on Blood lipid profiles of broilers at 35 day (Mean ± SD)

Parameters Groups	Cholesterol mg/dl	Triglyceride mg/dl	HDL mg/dl	VLDL mg/dl	LDL mg/dl
Group 1 (CON)	108.8 ± 2.3 C	78.0 ±4.3 B	74.2 ±2.6 A	15.0 ±0.8B	19.6 ±4.7C
Group 2 (AF)	136.8 ± 3.0 A	92.8 ±2.2 A	67.0 ± 3.0 B	18.5 ±0.4A	51.2 ±4.2A
Group 3 (AF+M)	122.2 ± 2.3 B	81.2 ± 0.8 B	71.4 ±3.1AB	16.2 ±0.2B	33.5 ±3.1B
Group 4 (AF+TP)	121.0 ± 5.1B	84.0 ± 1.0 B	75.2 ± 2.2 A	16.2± 0.2B	20.9 ±1.1C
Group 5 (TP)	110.8 ± 1.9 C	82.8 ± 4.6 B	72.2 ± 1.4 A	16.3 ±0.9B	22.2 ±2.6C

Difference letters in the same column represent a significant difference at (P<0.05)

4.6.5. Effect of TP on some antioxidant enzyme and MDA level of broilers at 28 day

A significant decreased (P<0.05) was noticed in the concentration of Gpx and SOD in the AF group compared with the other groups table (4-6-5). In addition, in the same

table the results showed a significant increase (P<0.05) in concentration of MDA in the AF group compared with other treated groups.

Table (4-6-5) Effect of TP on some antioxidant enzyme and MDA level of broilers at 28 day (Mean ± SD)

Parameters Group	SOD U/mL	GPX mmole/mL	MDA nmole/mL
Group 1(CON)	68.2 ± 0.8 A	20.1 ± 0.8 A	1.1 ± 0.01 B
Group 2 (AF)	55.1 ± 1.8 B	14.6 ± 1.1 B	2.1 ± 0.6 A
Group 3 (AF+M)	65.5 ± 1.1 A	19.7 ± 0.9 A	1.0 ± 0.02 B
Group 4 (AF+TP)	66.8 ± 2.7 A	19.8 ± 0.4 A	1.3 ± 0.4 B
Group 5 (TP)	67.7 ± 1.4 A	19.3 ± 0.7 A	1.2 ± 0.2 B

Difference letters in the same column represent a significant difference at (P<0.05)

4.6.6. Effect of TP on some antioxidant enzyme and MDA level of broilers at 35 day

A significant decreased (P<0.05) was noticed in the concentration of Gpx in the AF group compared with the other groups table (4-6-6). At the same time there was no significant difference at (P>0.05) in SOD and MDA among all groups.

Table (4-6-6) Effect of Tomato TP on some antioxidant enzyme and MDA level of broilers at 35 day (Mean ± SD)

Parameters Group	SOD U/mL	GPX mmole/mL	MDA nmole/mL

Group 1(CON)	74.19 ± 6.52 A	20.1 ± 1.0 A	1.08 ±0 .01 A
Group 2 (AF)	66.21 ± 3.20 A	15.2 ± 0.9 B	1.09 ±0 .02 A
Group 3 (AF+M)	68.42 ± 7.40 A	20.3 ± 0.7 A	1.09 ±0 .02 A
Group 4 (AF+TP)	67.64 ± 4.26 A	20.1 ± 0.6 A	1.08 ±0 .01 A
Group 5 (TP)	65.87 ± 6.46 A	19.1 ± 0.9 A	1.11 ±0 .01 A

Difference letters in the same column represent a significant difference at (P<0.05)

4.7. Residues of aflatoxins in the muscles of the breast and liver of chickens at the age of 28 and 35 for the second and fourth groups

Table (4-7) showed there were residues of aflatoxins in chicken breast muscles in day 28 (114.25) ppb and in day 35 (15.58) ppb in AF group and in chicken liver in day 28 (126.58) ppb and in day 35 (53.56) ppb. Also in same table noticed residues of aflatoxins in chicken breast muscles in day 28 (14.74) ppb and in day 35 (3.59) ppb in AF+TP group and in chicken liver in day 28 (20.15) ppb and in day 35 (11.23) ppb.

Table (4-7) Residues of aflatoxins in the muscles of the breast and liver of chickens at the age of 28 and 35

parameter Ages	breast muscles (AF)	breast muscles (AF+TP)	liver (AF)	Liver (AF+TP)
Day 28	114.25(ppb)	14.74 (ppb)	126.58(ppb)	20.15(ppb)
Day 35	15.58 (ppb)	3.59 (ppb)	53.56 (ppb)	11.23 (ppb)

4.8. Histopathological changes of liver

The results of the histopathologic examination of the liver are shown in figures (4-1) & (4-5) There was no visible lesion observed in livers of the broilers in control and (TP) groups. Livers from broilers consuming the (AF + TP) diet showed mild of inflammation and slightly portal vein congestion figure (4-3) and the same results in

Livers from broilers consuming the AF plus mycofix figure (4-4). Livers from broilers fed with the AF showed lesions perivascular lymphocyte infiltration in the parenchyma and the portal vein congestion. Hyperplasia of the bile duct also, necrosis of the hepatocytes and congestion figure (4-2).

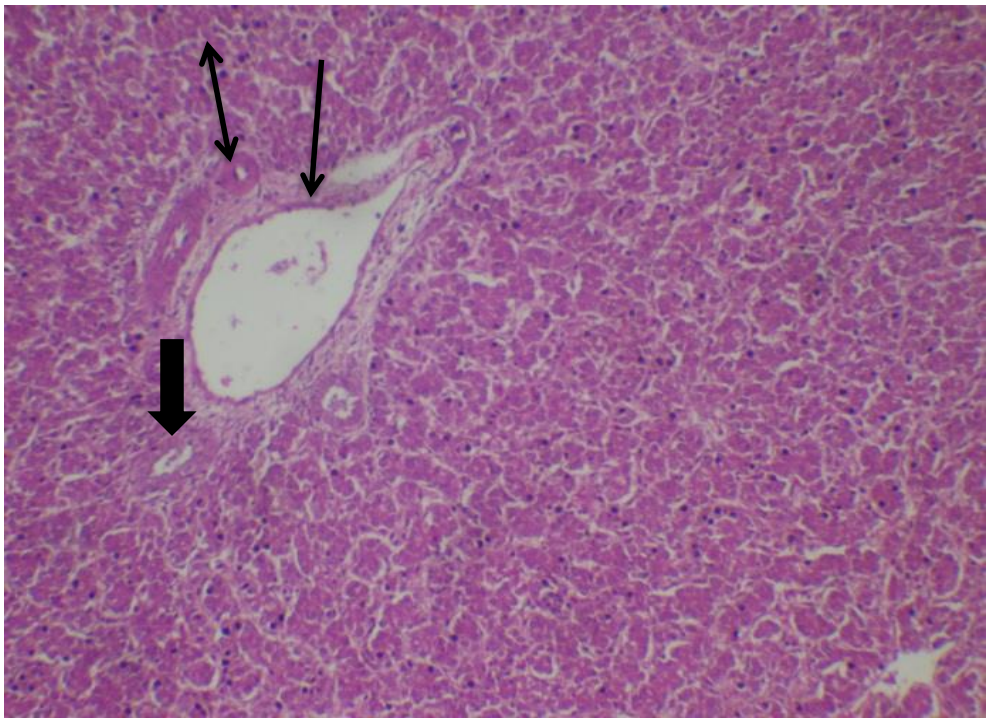

Figure 4-1: Section of the liver of chicken from the control group showing normal liver histology that devoid of any inflammation. Similar morphology was seen in the control animals throughout the experimental period: portral vein (thin arrow), bile duct (thick arrow), and hepatic artery (double arrow) H&E X100.

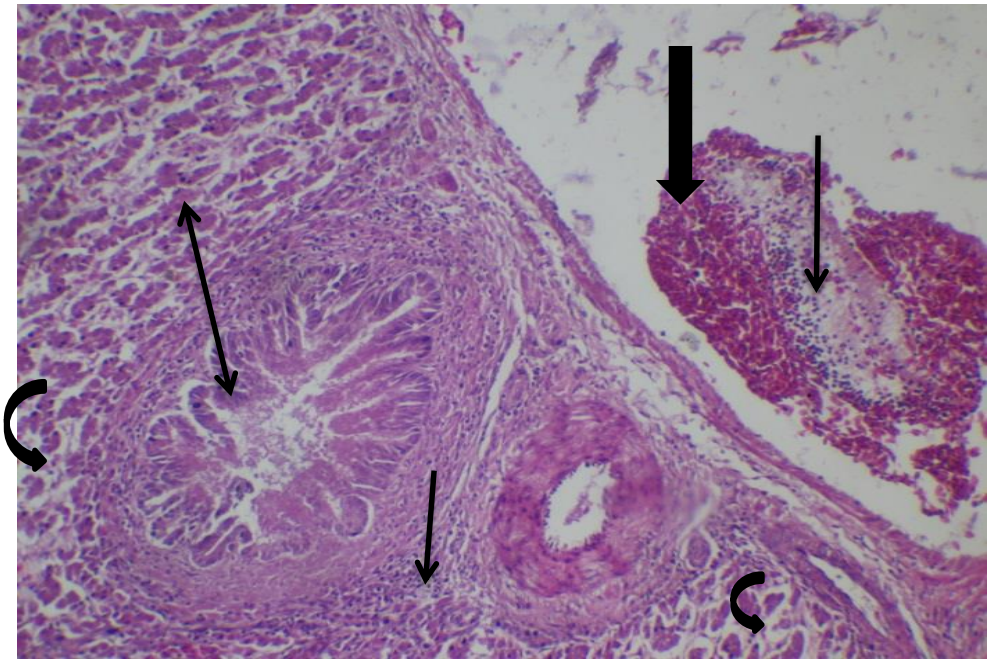

Figure 4-2: Section of the liver of chicken from the AF group necropsied at 28 days. Note a perivascular lymphocyte infiltration in the parenchyma (thick arrow) in and around the portal vein congestion (thin arrow) artery. Hyperplasia of the bile duct (double arrow) also, necrosis of the hepatocytes and congestion (carve arrow) H&E X100.

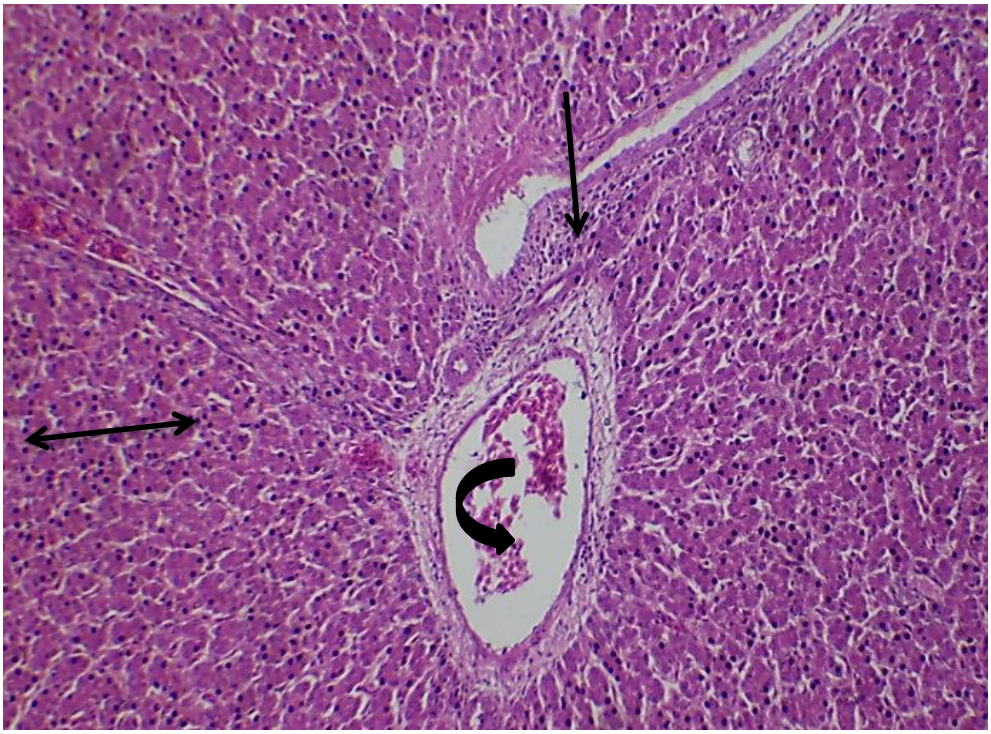

Figure 4-3: Section of the liver of chicken from the AF + TP group necropsied at 28 days. Showing normal liver histology that devoid mild of inflammation (some aggregation of lymphocytes) thin arrow, revealing by reduction in severity of inflammatory reaction with normal architecture (double arrow) with slightly portal vein congestion (carve arrow) H&E X100.

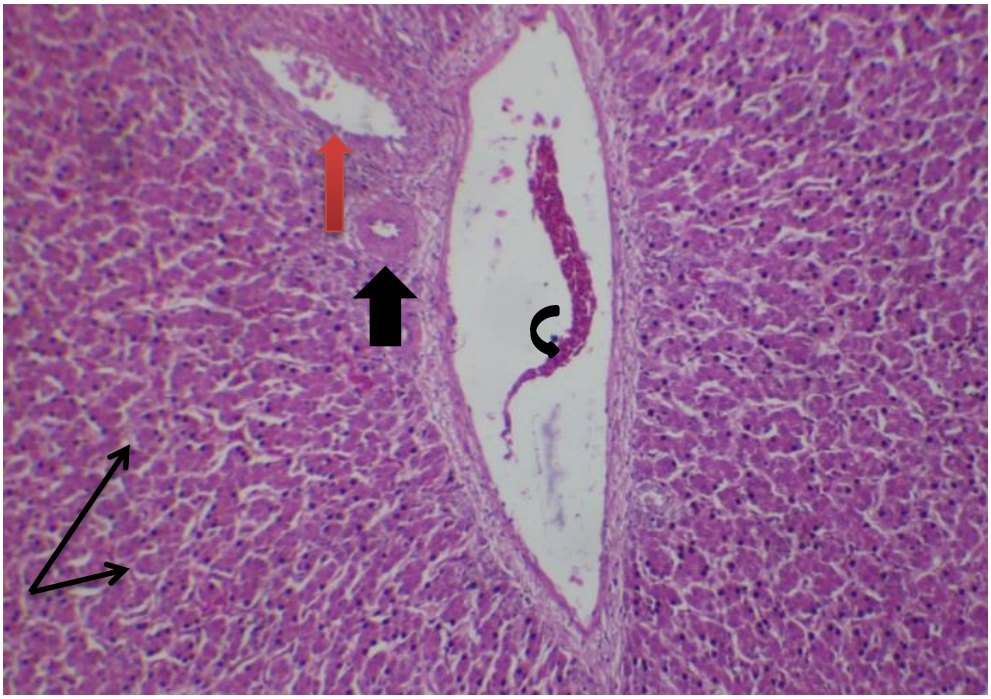

Figure 4-4: Section of the liver of chicken from the AF + M group necropsied at 28 days. Showing normal liver histology that devoid mild of inflammation, revealing by reduction in severity of inflammatory reaction with normal hepatocyte architecture (thin arrow) slightly portal vein congestion (carve arrow), portal artery (thick arrow) and bile duct (red arrow) H&E X100.

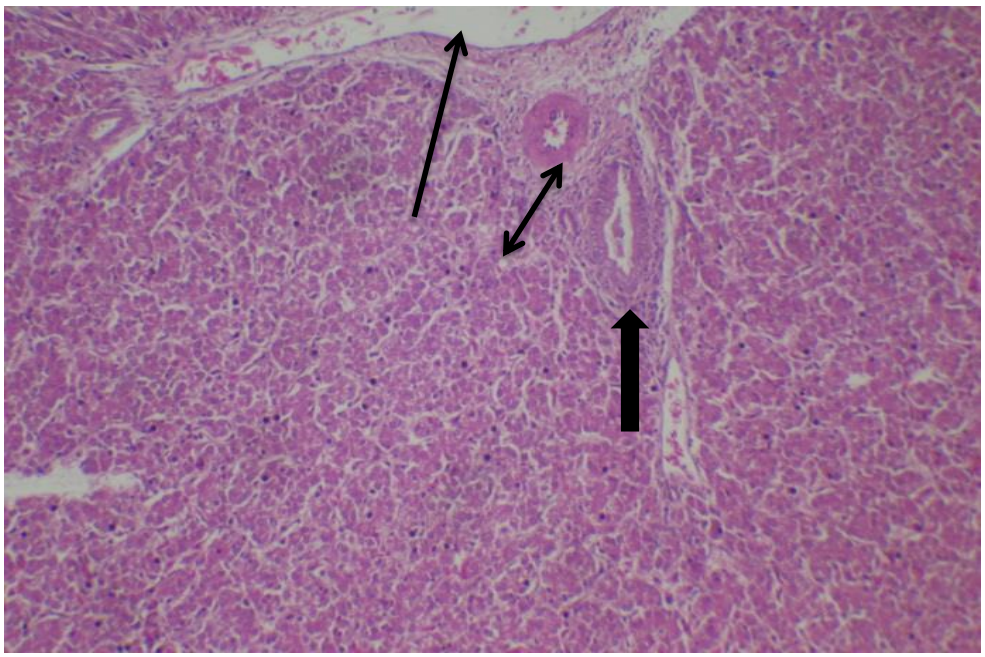

Figure 4-5: Section of the liver of chicken from the TP group necropsied at 28 days. showing normal liver histology that devoid of any inflammation. Similar morphology was seen in the control animals throughout the experimental period: portral vein (thin arrow), bile duct (thick arrow), and hepatic artery (double arrow) H&E X100.

Chapter Five: Discussion

5.Discussion

In this study, we aimed to demonstrate the effective role of tomato powder in minimizing the damage of broiler chickens exposed to contaminated diet with aflatoxin. Therefore, we will present and discuss the damage caused by the toxins and then the protective effect of the tomato powder in reducing those damages.

5.1 Growth performance

Results of the present study reflected that dietary aflatoxin adversely affected the body mass as well as performance of the broilers. Similar findings were also noticed earlier of broilers (Denli *et al*., 2004 ; Sakhare *et al*., 2007). AF significantly decreases BW gain, feed intake and impairs feed conversion rate (Oguz *et al*., 2000; Denli *et al*., 2009).

The present findings were in accordance with the similar reports like significant decrease in body mass, decline in feed intake and poor FCR in mycotoxin induced birds by (Hashem & Mohamed, 2009 ; Sawale *et al*., 2009). The adverse effects of AF on body mass, feed intake and FCR have been related with a decrease in the protein synthesis, energy utilization and reduced pancreatic digestive enzyme productions (Safameher, 2008) which results in a deterioration of the digestive and metabolic efficiency of the birds. In the present study, decrease in body weight may be due reduced feed consumption and feed convertion, Additionally reduced weight gain in induced birds is due to depressed appetite and impaired nutrient utilization (Manegar *et al*., 2010).

The use of plant rich polyphenols seem to be a promising strategy for improving products quality. In dietary manipulations, antioxidants are introduced into the muscle via the animal feed. Various authors have reported that inclusion of natural antioxidants in animal diets not only slows down oxidation, but also greatly improves meat quality when compared to diets with no antioxidants (Brenes *et al*., 2016).

Tomato contains other carotenoids, including phytoene, phytofluene, and the provitamin A carotenoid β-carotene, which may have a synergistic effect with lycopene, it improved the performance, namely live weight gain and feed conversion was in agreement with results of (Rao & Agarwal, 1999).

5.2 Weight of liver, spleen, thymus and bursa Fabricius

The present study revealed that there was a significant increase ($P<0.05$) in the weight of liver in the AF group respectively compared with the other treated groups, in the same table the results showed a significant decrease ($P<0.05$) in the weights of the spleen, thymus and bursa in the second AF group compared with other treated groups (table 4-3-1). There was in agreement with results of (Valchev *et al*., 2017 ; Nemati *et al*., 2015 ; Shareef & Sito, 2019). Aflatoxin have adverse effects on the weight of the liver. Similar findings were reported by (Kermanshahi *et al*., 2007) that enlargement and discoloration of the liver were associated with aflatoxin. whereas aflatoxin increase liver weight reported that diet contaminated with toxins results in an adverse effect on liver morphology too (Verma *et al*., 2004 ; Che *et al*., 2010). Contamination of feed with mycotoxins increases lipid deposition in the liver and weight increases (Sharlin *et al*., 1980). Also Quezada *et al*., (2000) ; Magnoli *et al*., (2011) reported that AF affects the liver in the form of increased fat and relative weight and a reduction of the overall secretory capacity of the organ.

On the other hand, the addition of AF to the diet suppressed the relative weights of the lymphoid organs weight loss for spleen, thymus and bursa, respectively. These results align with the findings of Solcan *et al*., (2014) ; Attia *et al*. (2016), who showed that diet contamination with AF induced a marked and progressive thymocytes depletion via the apoptotic process and reduced the relative weights of the bursa, spleen and thymus. Campbell *et al*., (1983) found that the relative weight of the bursa of Fabricius, spleen and thymus its follicle count were reduced by AF. Relative weight of the bursa of Fabricius, spleen and thymus were significantly reduced, depending on the dose of AF (Thaxton *et al*., 1974). Thus, the observations in the present study that the reduced relative weights of the spleen, thymus, and bursa of Fabricius may be due to the detrimental effect of AF on the immune system.

Indeed carotenoids reduced the toxic effects of aflatoxin with respect to the relative weights of the immune organs (Cheng *et al*., 2001). lycopene may affect the levels of cytokine and therefore can influence the immune responses and inflammation (Palozza *et al*. 2011).

5.3 Hepatosomatic Index

The results of table (4-3-3) showed a significant increase in liver index in the second group (AF) compared with other treated groups.That was similar to the results from other researchers (Miazzo *et al*., 2000 ; Sakhare *et al*., 2007 ; Safameher,(2008) hypothesize that feeding aflatoxins resulted in the inhibition of hepatic protein synthesis and lipid metabolism, including a decrease in hepatic protein content, the formation of fatty liver, and an increase in the relative weight of livers. Birds fed corn naturally contaminated with AF tended to have an increase in the relative weight of livers. The inhibition of lipid transport may result in lipid accumulation and the formation of a specific and enlarged fatty liver (Yang *et al*., 2012).

Results in the same table (4-3-3) showed significant decrease in liver index in the AF&TP group the present study it was noted that there was protective role for tomato powder in recovery of hepatocytes compared with group exposed to AF only and tomato powder caused healing of hepatic injury induced by AF , that was further confirmed by the histological section of liver which showed normal architecture, normal hepatocyte and normal central vein (Elvira-Torales *et al*., 2019).

5.4 Sensory traits

The present study revealed that there was significant increase ($P<0.05$) noticed in the sensory traits of tenderness, juicy, flavor, color and palatability in the AF&TP, and TP groups in (table 4-4). These an agreement with results of (Adeyemi & Olorunsanya, 2012). Where Sensory results showed that tomato powder improved the tenderness, juicy, color, flavor and palatability of broiler meat. Also according to (Itankar *et al*., 2010). Organoleptic evaluation revealed that the average scores for tenderness, juicy, flavor, color and palatability of broiler meat were declined with AF group.

5.5 Immunity Parameters

The ND and IBD antibody titer level showed significant decrease table (4-5-1,4-5-2) in second group (AF) that broilers exposed to contaminated diet with aflatoxin. This may be associated with that aflatoxins works as an inhibitor of protein synthesis and as resulted, destroy cells and tissues with a high protein turnover such as that found in the liver, immune system or gut epithelium, which is most

susceptible to the toxic effects of AF. In this way, exposure to AF has been demonstrated to suppress the immune response in poultry (Hossein & Gurbuz, 2015). Furthermore AF can inhibit the development of the thymus gland or effect the relative weight of the bursa of Fabricius, which may result in serious deficiencies in both cellular and antibody responsiveness of the chicken immune system (Celik *et al*., 2000). Inhibition of macrophage functions, T lymphocyte activity or cytokine expression by AF results in vaccine failure or pathogen persistence, as exemplified in many studies by reduced immunoglobulin production (Yunus *et al*., 2008).

Recent epidemiological data indicate a high correlation between outbreaks of Newcastle disease and AF contamination of broiler rations (Yunus *et al*., 2008). A variety of factors such as vaccination failure, infection by immune suppressive diseases, and abuse of antibiotics can induce immunodeficiency (Hashemipour *et al*., 2013).

Table (4-5-1 ,4-5-2) revealed significant increase in ND &IBD antibody titer level in forth group that broilers fed on T&AF compared with AF group. Herbs that are rich in flavonoids extend the activity of vitamin C, act as antioxidants, and may therefore enhance immune function (Acamovic & Brooker, 2005). This is in agreement with the previous ports of improving antibody response caused by herbal depravities (Vahid *et al*., 2012 ; Hashemipour *et al*., 2013). In poultry production, it is more effective to use immune stimulators to improve immunity and decrease susceptibility to infectious disease (Ebrahimzadeh *et al*., 2018). Lycopene mediates cell growth regulation, immune response, and modulation of phase I and II detoxifying enzymes and gene transcription (Palozza *et al*., 2011). Effect of lycopene in immune system modulation is not as simple as absorption and execution and it could be a result from a complicated network consisted of simultaneous activation of various immunomodulatory pathways (Manabe *et al*., 2014). Changes in lymphocyte concentration could be an indicator of activation of cell-mediated or humoral immune response and thus, lycopene was proposed to be able to influence immune response and the production of antigen-specific antibodies (Puah *et al*., 2021).

5.6 Biochemical parameters

5.6.1 Liver and kidney enzymes

The significant increased in the level of liver enzymes AST & ALT table (4-6-1) in second group (AF) that exposed to contaminated diet with aflatoxin was in agreement with results of (Denli *et al*., 2009 ; Naseem *et al*., 2018). Liver is the primary organ of AF accumulation and metabolism, liver is also the main site where AF is metabolized and where the metabolites bind with nucleic acids and proteins (Yunus *et al*., 2011). ALT is particularly useful in measuring hepatic necrosis, as it is a key cytoplasmic enzyme present in liver and other cells. The enhanced serum levels of AST and ALT have been attributed to the damaged structural integrity of the liver, because they are cytoplasmic in location and subsequently liberated into circulation after cellular damages (Ahsan *et al*., 2009), So, AST and ALT are increased level in the blood indicates cellular damage and increased membrane permeability and their altered metabolism (Ramazzotto &Carlin, 1978).

Manning & Wyatt, (1990) reported that AST was elevated in aflatoxin intoxicated birds compared to controls. The liver is the main organ that processes mycotoxins, detoxifies them, and protects the body against their toxic effects. The liver also is a central organ for lipid, protein, and amino acid metabolism, and their utilization (Fouad & El-Senousey, 2014 ; Fouad *et al.,* 2019). Abnormal liver size may be associated with liver malfunctions. AF can cause imbalanced lipid metabolism, promoting lipid deposition in the enlarged liver (Siloto *et al.,* 2013), with repressing the activity of antioxidant enzymes and anti-inflammatory cytokines, enhance lipid peroxidation, pro-inflammatory cytokines and increase hepatocyte apoptosis (Muhammad *et al.,* 2018 ; Wang *et al.,* 2019). The usual deleterious effects of AF on hepatocytes result in high concentrations of ALT and AST in poultry blood after feeding diets containing AF (He *et al.,* 2013 ; Gómez-Espinosa *et al.,* 2017; Muhammad *et al.,* 2018). AST (found in mitochondria) and ALT (found in the cytoplasm) are involved in hepatic protein metabolism, and they can determine the cell integrity (Jiang *et al.,* 2014).

Hepatotoxicity mechanism caused by AF occurs by reducing GPX and SOD that results in the formation of radical oxygen species (ROS). ROS that are not balanced with antioxidants and can cause oxidative stress (Wang *et al*., 2005). Free radicals will damage the cell membrane, mitochondria, and endoplasmic reticulum. This condition causes activation of enzymes phospholipase, protease, endonuclease, and ATPase which results in decrease the phospholipids, disruption of membrane

proteins, DNA fragmentation, and decreased ATP that causes necrosis in hepatocytes (Sulistyowati *et al*., 2013). Tomato powder that rich in lycopene has high antioxidant activity. Antioxidants work by giving electrons to stop reaction of free radicals so the oxidation of fats and proteins could not be continued, thus cell damage can be prevented (Purnama *et al*., 2020).

There is a significant decrease in AST and ALT concentration in group treated with TP in comparison with AF groups and that present result agrees with (Ţigu *et al*., 2016). There is strong evidence that lycopene (main component of tomato) is capable of reducing serum ALT and AST (Eze *et al*., 2016).

The present findings of significantly elevated serum creatinine were in agreement with those of (Hashem & Mohamed 2009 ; Rashidi *et al*., 2018). Increased level of creatinine may be attributed to the accelerated rate of protein catabolism and nephrotoxic effect of aflatoxin evident by pathomorphological changes in kidneys. The kidney is responsible for maintaining extracellular medium homeostasis, electrolyte balance, as well as removing the products of body metabolism (Eaton & Pooler, 2004). Kidneys also take part in detoxification of aflatoxins and are also among the organs where most of the aflatoxin residues are detected (Fernandez *et al*., 1994).

Kidneys represent the major control system maintaining body homeostasis. The plasma concentrations of creatinine determine renal function therefor consider as biomarkers for kidney disease (Levey *et al*., 1999). The mechanism underlying proanthocyanidins nephroprotection may be due to the marked radical scavenging ability of proanthocyanidins (Sato *et al*., 2005). These effects of lycopene are associated with the prevention of the oxidative stress, which increased the antioxidant capacity of the body and maintenance of the permeability of the cell membrane (Yilmaz *et al*., 2018).

5.6.2 lipid profiles

The results of the current study showed a significant decrease ($P<0.05$) was noticed in the serum concentration of cholesterol, triglyceride, LDL and VLDL at the AF&TP, AF&M and TP groups respectively compared with the AF group, while HDL was increased significantly ($P<0.05$) in the same groups (table 4-6-3).

Tomato powder (lycopene content) as an antioxidant source was expected could decrease the total cholesterol level of broiler chickens exposed to aflatoxin. The

mechanisms of which antioxidant decreasing cholesterol were : 1) inhibit the activity of HMG-CoA reductase to decrease mevalonate synthesis as a basis of cholesterol formation; 2) Inhibit the Cholesterol Acil Transferase which will reduce the storage of ester cholesterol in the tissue; 3) increase the activity of LDL receptor to decrease LDL cholesterol in blood (Palozza *et al*., 2012).

5.6.3 Antioxidant enzymes

The present study revealed that there was significant increase in MDA level in table (4-6-5) and significant decrease in Gpx and SOD enzymes in AF group that broilers exposed to contaminated diet with aflatoxin and these results were matched with previous studies (Sharma *et al*., 2011; Gowda *et al*., 2009).

Firstly can be defined antioxidant as "any substance that, when present in low concentrations compared to that of an oxidizable substrate, delays or inhibits the oxidation of that substrate" (Chauhan *et al*., 2016). Antioxidants may protect cells from the damage caused by free radicals. Antioxidant nutrients and enzyme defenses are fundamental protectors against all forms of stress (Ojha *et al*., 2010).

In animals, oxidative stress may occur as a consequence of nutrition, including the contamination of feed with fungal toxins (Georgieva *et al*., 2006 ; Frankic *et al*., 2008). Aflatoxin induced lethally injured hepatocytes in an unmanageable disorder, which results in mitochondrial dysfunction, enzyme inhibition and denaturation of structural proteins. the increase in hepatic MDA level may be attributed to the fact that AF is metabolized by the cellular cytochrome P450 enzyme system (Kheir Eldin *et al*., 2008), this leads to lipid peroxidation and cellular injury (Stresser *et al*., 1994) also may be due to significant reduction in the activities of enzymatic antioxidants like Gpx in level which is observed in present study. The SOD and Gpx as the key enzymes of antioxidant system can scavenge free radicals generated from oxidant stress, reduce oxidative damage and maintain cell structure (Chen *et al*., 2014).

On the other hand, the results showed significant decreased in MAD level and increase in Gpx and SOD enzymes in table (4-6-5) in AF & TP group.

Findings that serum MDA levels were significantly lowered after lycopene treatment. Additionally, Alshatwi *et al*., (2010) reported that tomatoes antioxidants might contribute to the protection against peroxidation. Diminishing MDA levels in this study also might have been attributed to lycopene which protects cells against oxidative stress. Similar observations have been made by Heber & Lu, (2002) who

attributed the ability of lycopene in quenching free radical anions and increasing the number of conjugated double-bonds. It was found that TP supplementation elevated the values of SOD, Gpx, which are important enzymes associated with the high antioxidant activity, subsequently, decreasing oxidative stress properties like antioxidant, antiviral, inflammatory and immunomodulatory. These findings agreed with those of (Genaro-Mattos *et al*., 2015 ; Elwan *et al*., 2019).

The activity of antioxidant enzymes could induce as a result to the oxidative stress or could diminish through direct or indirect action of the mycotoxins (Yilmaz *et al*., 2017). Lycopene acts as an antioxidant by virtue of its conjugated p-electron system, which can react with oxygen radical species such as peroxy and hydroxy radicals as well as non-radical species such as ozone and H2O2 (Halliwell & Gutteridge, 2015). Lycopene has a robust antioxidant defense system, attributed to its acyclic structure, numerous conjugated double bonds, and high hydrophobicity, thus prevents the onset of carcinogenesis and atherogenesis processes by protecting/stabilizing biomolecules such as DNA, proteins, lipids, and lipoproteins. Lycopene, as the main carotenoid in tomato products, possesses the greatest ability to quench singlet oxygen compared to the other carotenoids. It also scavenges the free radicals via three difference mechanisms: adduct formation, electron transfer, and hydrogen atom transfer reported that lycopene and torulene are more reactive scavengers of peroxide radicals than β-carotene (Yilmaz *et al*., 2017).

The lycopene effects are related to the prevention of the oxidative stress and increased the antioxidant capacity of the body , maintenance of the permeability of the cell membrane (Yilmaz *et al*., 2018), and able to reduce not only the oxidative damage of DNA to a considerable extent but also is effective in hindering the lipid peroxidation process (Matos *et al*., 2000 ; Yilmaz et al., 2006).

5.7 Histopathological study of Liver

The liver is the main target organ for the metabolism and detoxification of aflatoxins, and histopathological examination by optical microscope can be used as an effective method for diagnosis of aflatoxicosis (Ellakany *et al*., 2011). Previous studies have demonstrated the hepatotoxic effect of AF on broilers, leading to pathological lesions of the livers (Eraslan *et al*., 2006 ; Kumar & Balachandran, 2009). Aflatoxins can also cause important gross and microscopic changes in the

liver, such as hepatomegaly, paleness, hydropic degeneration, fatty change, bile-duct hyperplasia, and periportal fibrosis (Ortatatli & Oğuz, 2001). Aflatoxin is mainly metabolized in the liver and its second metabolite of AF- 8,9-expoxide can combine with DNA, cause canceration of hepatic cells, and damage the hepatic functions there after (Yoshizawa *et al*., 1982 ; Eaton & Gallagher, 1994).

The present study noted that there was protective role for tomato powder in recovery of hepatocytes compared with group exposed to AF only and tomato powder caused healing of hepatic injury induced by AF, that was further confirmed by the histological section of liver which showed look like normal architecture, normal hepatocyte and normal central vein (Elvira-Torales *et al*., 2019). Antioxidants can advance to increase the defense systems of cells, for this reason, these antioxidants are necessary to cope with excessive ROS production (Baş *et al.*, 2016). Lycopene becomes protective towards the examined parameters because of its antioxidant properties, It may be an indirect scavenger of ROS or it can boost antioxidant enzyme activities.

Chapter Six: Conclusions and Recommendations

6.1 Conclusions

Based on the current research findings, the conclusions are the following

1. Tomato powder (10 mg/kg) can be used as a feed supplement in broiler chickens from 1 to 35 days of age to enhance their antioxidant status.

2. Tomato powder caused healing of hepatic injury induced by AF.

3. Tomato powder can help in minimizing damage to the liver exposed to mycotoxins.

4. Addition TP leading to decrease LDL and increase HDL.

5. Addition TP leading to improve sensory traits.

6. Tomato powder improve BW, FCR, FI.

6.2 Recommendations

1. Much more studies should be done to use the effect of raw tomatoes in layer.

2. Studying the effect of TP on other stress.

3. A future study to investigate the possibility of TP inclusion as a supplement in the drinking water during periods of stress, vaccinations, and the acceptance rate of broiler chickens to consume TP supplemented water.

4. Studying the effect of TP on other parameters such as hormones.

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Appendices

Appendix: 1

1- Materials and Procedure of determination aflatoxin by ELISA test. ELISA Kit (Neogen® Veratox®)

Materials provided

1. 48 antibody-coated microwells.
2. 48 red-marked mixing wells.
3. 4 yellow-labeled bottles of 0, 5, 15 and 50 ppb aflatoxin controls (see precautions for handling of methanol solution).
4. 1 blue-labeled bottle of aflatoxin-HRP conjugate solution.
5. 1 green-labeled bottle of K-Blue® Substrate solution.
6. 1 red-labeled bottle of Red Stop solution.

Materials required

1. Extraction materials (items 3 through 5 available in kit form):
    1. 70% ACS Grade methanol.
    2. 250 mL graduated cylinder.
    3. Container with 125 mL capacity.
    4. Neogen filter syringes, Whatman #1 filter paper, or equivalent.
    5. Sample collection tubes.
2. High-speed blender.
3. Agri-Grind grinder or equivalent.
4. Scale capable of weighing 5-50 grams.
5. Microwell reader with a 650 nm filter.
6. Pipettor, 12-channel.
7. Pipettor, 100 µL.
8. Pipette tips for 100 µL and 12-channel pipettors.
9. Paper towels or equivalent absorbent material.
10. Plastic bucket for use as waste receptacle.
11. Microwell holder.
12. Timer.
13. Waterproof marker.
14. Wash bottle.
15. 2 reagent boats for 12-channel pipettor.

16. Distilled or deionized water.

Procedure :

1. Add 100 µL conjugate to red marked mixing wells.

2. Add 100 µL controls and samples to the red marked mixing wells.

3. Mix. Transfer 100 µL to antibody wells. Incubate for 2 minutes.

4. Dump liquid from antibody wells.

5. Wash wells thoroughly 5 times with deionized water.

6. Tap out water on absorbent paper towel.

7. Transfer 100 µL substrate from reagent boat to antibody wells using 12-channel pipettor. Incubate for 3 minutes.

8. Transfer 100 µL Red Stop from reagent boat to antibody wells.

9. Read results using a microwell reader with a 650 nm filter.

Appendix: 2

Procedure of determination immunity of ND and IBD by ELISA test

ELISA Kit (Synbiotics–USA)

The procedure used in this test was performed according to the manufacturer instructions listed in the ProFLOK® (ND and IBD) ELISA Kit (Synbiotics–USA), which is a rapid serologic test for the detection of IBV antibody in chicken serum samples. It was developed primarily to aid in the detection of pre and post-vaccination (ND and IBD) antibody levels in chickens.

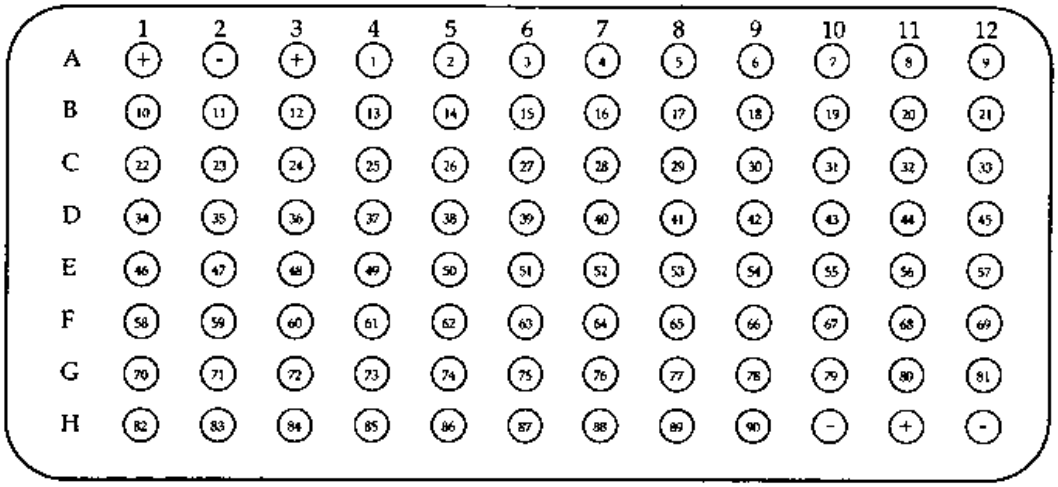

Figure Diagram explain microtitre plate test kit

ELISA was performed as the following:-

Each test sample 1:500 was diluted by adding 1 µl to 0.5 ml of sample diluents.

1. Fifty µl of diluents was added to each plate that contained coated antigen.
2. Fifty µl of positive control was added in wells (H11, A3, and A1) for the plate.
3. Fifty µl of negative controls was added in wells (A2, A10, and H12) for the plate.
4. Fifty µl of diluted serum was added to the suitable wells for the presided plate.
5. Plate was incubated for 30 minutes at room temperature.

6. Plate holes were washed by using washing solution.
7. Hundred µl of diluted antiserum was added at ratio of 1:100 and conjugated with (horseradish peroxides) for each hole.
8. Plate was incubated for 30 minutes at room temperature.
9. All the plate holes were washed by using washing solution in amount of 300 µl twice with good drying.
10. Hundred µl of substrate was added to expose the color, and then the plates were left at room temperature in a dark place.
11. Hundred µl of stop solution (H_2SO_4) was added.
12. Results were read through recording the optical density absorbance of the control and the samples antibody titers were calculated automatically, by using ELISA reader using profile flock software.

Appendix: 3

1-(AST)Serum Aspartate aminotransferase activity AST is determined by using a special kit (SPECTRUM AST – kit, Egypt)(Young,1990)

Principle:

Colorimetric determination of AST activity is obtained according to the following reactions:

AST

Aspartate+αketoglutarate $\xrightarrow{\text{AST}}$ oxaloacetate+glutamate

The reaction

The oxaloacetate formed is measured in its derivative form, 2,4-dinitrophenylhydrazone.

Reagents:

Reagent1A **ST**	Phosphate buffer pH7.5 L-Aspartate 2-Oxoglutarate Sodium Hydroxide Sodium Azide	100mmol/l 100mmol/l 5 mmol / l 140mmol/l 12mmol/l
Reagent2 **Color** **Reagent**	2.4dinitrophenylhydr azine HCL	2mmol /l 8.4 %

Procedure

Wave length: 546 nm (530 – 550 nm) Zero adjustment: reagent blank:

Pipette into test tubes:

Appendices

Reagent	Reagntb lank	Sample
Reagent(Buffer)	0.5ml	0.5ml
Sample	-------	100µl
Distilled water	100µl	------

Mix and incubate for exactly 30 minutes at 37Cº

Reagent 2	0.5ml	0.5ml

Mix and incubate for exactly 20 minutes at 20-25Cº

Sodium hydroxide	5ml	5ml

Mix and measure absorbance of specimen against reagent blank at 546nmafter 5 minutes

Appendices

Calculation

Obtain the AST activity from the following table.

Absorbance	Value of ASTU/L	Absorbance	Value of ASTU/L
0.020	7	0.100	36
0.030	10	0.110	41
0.040	13	0.120	47
0.050	16	0.130	52
0.060	19	0.140	59
0.0170	23	0.150	67
0.080	27	0.160	76
0.090	31	0.170	89

Linearity:

The assay is linear up to 89U/L .if the absorbance exceeds 0.170 at 546nm, sample should be diluted 1+9 using sodium chloride and repeat the assay (result x10).

Appendices

2-(ALT)Serum Alanine aminotransferase activity ALT is determined by using a special kit(SPECTRUMALT–kit,Egypt)(Young,1990).

Principle

Colorimetric determination of ALT activity is obtained according to the following reactions:

Alanine+αketoglutarate $\xrightarrow{\text{ALT}}$ pyruvate+glutamate

The reaction:

The pyruvate formed is measured in its derivative form, 2,4-dinitrophenylhydrazone

Reagents

Reagen 1 **ALT**	Phosphate buffer pH7.5 D-Alanine 2-OxoglutarateSodium Azide	100mmol/l 200mmol/l 6mmol/l 12mmol/l
Reagen2 **Color reagent**	2.4dinitrophenylhydrazine	2mmol/l

Procedure

Wave length: 546 nm (530 – 550nm) Zero adjustment: reagent blank:

Pipette into test tubes:

Appendices

Reagent	Reagentblank	Sample
Reagent(Buffer)	0.5ml	0.5ml
Sample	-------	100µl
Distilled water	100µl	------

Mix and incubate for exactly 30 minutes at 37Cº

Reagent2	0.5ml	0.5ml

Mix and incubate for exactly 20 minutes at 20-25Cº

Sodiumhyd roxide	5ml	5ml

Mix and measure absorbance of specimen against reagent blank at 546 nm after 5 minutes.

Appendices

Calculation

Obtain the ALT activity from the following table

Absorbance	Value of ALTU/L	Absorbance	Value of ALTU/L
0.025	4	0.275	48
0.050	8	0.300	52
0.075	12	0.325	57
0.100	17	0.350	62
0.125	21	0.375	67
0.150	25	0.400	72
0.175	29	0.425	77
0.200	34	0.450	83
0.225	39	0.475	88
0.250	43	0.500	94

Linearity: the assay is linear up to 94 U/L .if the absorbance exceeds 0.5 at 546nm, sample should be diluted 1+9 using sodium chloride and repeat the assay(resultx10).

Appendix: 4

1- Creatinine

Assay Principle

Creatinine reacts with picric acid under alkaline condition to form a yellow-red complex. The absorbance of the color produced, measured at a wavelength 492 nm, is directly proportional to creatinine concentration in the sample.

$$\textit{Creatinine + picrate} \xrightarrow{\textit{Alkaline pH}} \textit{= yellow-red complex}$$

***Reagents**

Standard (ST)

2 mg/dl 177 mmol/l

***Reagent 1 (R1)**

Picric acid 25 mmol/l

Surfactants

Creatinine Picric Acid Reagent contains a low concentration of picric acid, a chemical which, in its dry form, is flammable and potentially explosive. For this reason, it is recommended that drains be well flushed with water when disposing the reagent, spills be cleaned up at once, and avoid dryness of the material around the reagent bottle opening.

***Reagent 2 (R2)**

Sodium hydroxide 0.4 mol/l

Procedure

Pipette into test tubes
Working solution 1.0 ml
Standard or Specimen 100 µl

Mix, and after 30 seconds. read the absorbance A1 of the standard or specimen. After exactly 2 minutes later, read absorbance A2 of standard or specimen.

Calculation

A2 – A1 = Aspecimen or Astandard.

Concentration of creatinine in serum: $\textbf{\textit{Creatinine (mg/dL)}} = \frac{\textbf{\textit{A specimen}}}{\textbf{\textit{A standerd}}} \textbf{\textit{ x 2}}$

Appendix: 5

1-Measurement of Malondialdehyde (MDA):

The level of malondialdehyde was determined by a modified procedure described by Guidet and Shah (1989).The principle of the test was based on the reaction of MDA with thiobarbituric acid (TBA); forming an MDA-TBA_2 product that absorbs strongly at 532 nm as the following reaction.

Malondialdehyde + 2 TBA ⟹ Red complex

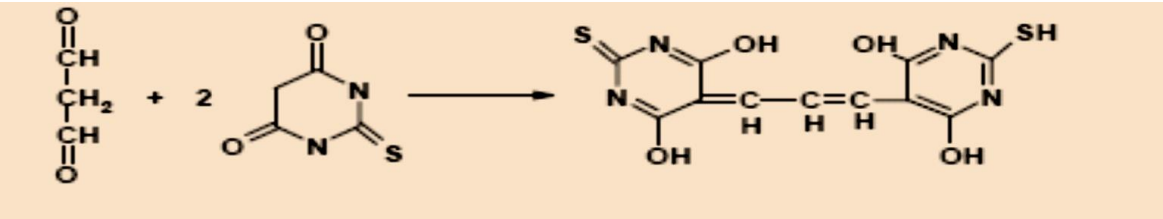

Figure: MDA assay reaction

Preparation of Reagents:

1. TCA reagent (70 %): Seventy grams of trichloroacetic acid (TCA) was taken and dissolved in a final volume of 100 ml of DW.

2. TCA reagent (17.5 %): Five milliliters of 70% TCA was taken and the volume was completed to 20 ml with DW.

3. TBA reagent (0.6 %):0.06 g of TBA was dissolved in a final volume of 10 ml of DW using a water bath for complete dissolving of TBA.

Procedure:

1. One hundred µL of serum sample was poured in a test tube and 1 ml of 17.5 % TCA was added.

2. One milliliter of 0.6 % TBA was added.

3. The tubes were mixed well by vortex, incubated in boiling water bath for 15 minutes, and then allowed to cool.

4. One milliliter of 70 % TCA was added.

5. The mixture was left to stand at room temperature for 20 minutes.

6. The tubes were centrifuged at 2000 xg for 15 minutes, and the supernatant was taken out for measuring spectrophotometrically at 532 nm.

2-Measurement of Serum Glutathione Peroxidase (Gpx):

Measurement of Serum Gpx was done by using a sandwich enzyme immunoassay Elisa kit. The microtiter plate used in this kit has been pre-coated with an antibody specific to GPX_1. Then, standards or samples were added to the right microtiter plate wells with a biotin-conjugated polyclonal antibody preparation specific for GPX. Afterwards, Avidin conjugated to Horseradish Peroxidase (HRP) was added to each microplate well and incubated. A TMB substrate solution was then added to each well. Only those wells that include GPX, biotin-conjugated antibody and enzyme-conjugated Avidin showed a change in color. The enzyme-substrate response was terminated by adding a sulphuric acid solution and the color change was measured spectrophotometrically at a wavelength of 450 nm. The concentration of GPX in the samples was then determined by comparing the O.D. of the samples to the standard curve

Reagent Preparation

1. All kit components and samples were brought to room temperature (18-25) before use.

2. Standard was reconstituted with 1.0 ml of standard diluent. It was left for 10 minutes at room temperature and shaken slowly (not to foam). The concentration of the standard in the stock solution was 600 U/mL the stock standard solution was used to produce a dilution series (figure 2-4). Each tube was mixed thoroughly before the next transfer. Seven points of diluted standard were setted up such as 600 U/mL, 300 U/mL, 150 U/mL, 75 U/mL, 37.5 U/mL, 18.75 U/mL, 9.38 U/mL, and the last EP tubes with Standard Diluent was the blank as 0 U/mL .

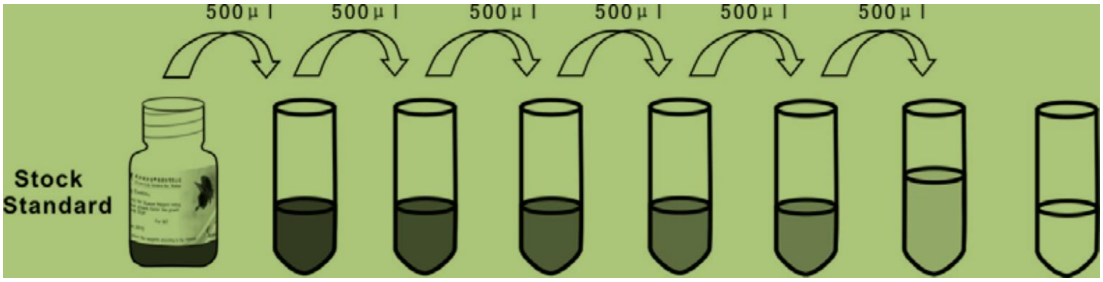

Figure: Protocol of working standard preparation of Gpx

Appendices

3.Assay Diluent A and Assay Diluent B **:** A mount of 6mL of Assay Diluent A or B Concentrate(2) was diluted with 6ml of deionized or distilled water to prepare 12 mL of Assay Diluent A or B. The prepared working dilution was not able to frozen.

4. Detection Reagent A and Detection Reagent B :The stock Detection A and Detection B were spined briefly or centrifuged before use. It was diluted to the working concentration with working Assay Diluent A or B, respectively (1:100).

5. Wash Solution - 20mL of Wash Solution Concentrate (30) was Diluted with 580ml of deionized or distilled water to prepare 600 mL of Wash Solution(1).

6. TMB substrate - The needed dosage of the solution was aspirated with sterilized tips and do not throw the leftover solution was not thrown into the vial again.

Assay Procedure

1. Wells were determined for diluted standard, blank and sample. 7 wells were prepared for standard 1 well for blank. Add 100 of each dilutions of standard (see Reagent Preparation), blank and samples were placed into the appropriate wells, Covered with the Plate sealer, and incubated for 2 hours at (37 ° C).

2. The liquid of each well was removed (without washing).

3. A mount of 100 **µl** of Detection Reagent A working solution was added to each well. Incubated for 1 hour at (37 ° C) after wrapping it with the Plate sealer.

4. The solution was Aspirated and washed with 400 of 1X Wash Solution to each well by using a squirt bottle, multi-channel pipette, manifold dispenser or auto washer, and it was let for 1~2 minutes. The remaining liquid was removed from all wells totally by snapping the plate onto absorbent paper (3 times). After the last wash, any remaining Wash Buffer was removed by aspirating or decanting. Then, the plate was inverted and blotted against an absorbent paper.

5. A mount of 100 **µl** of Detection Reagent B working solution was added to each well, incubated for 30 minutes at (37 ° C) after covering it with the Plate sealer.

6. The aspiration wash process was repeated for five times as conducted in step 4.

7. A mount 90 **µl** of Substrate Solution was added to each well. The plate was covered with a new plate sealer. Incubated for 15- 25 minutes at (37 ° C) (without exceeding 30 minutes), and protected from light. The liquid turned blue by the addition of Substrate Solution.

8. A mount of 50 **µl** of Stop Solution was added to each well. The liquid turned yellow by the addition of Stop solution. By tapping the plate's side, the liquid was mixed. If color change did not show uniform, the plate was gently tapped to ensure thorough mixing.

Calculation of Results

The average was taken for the same readings for each standard, control and samples, and the average zero standard optical density was subtracted. A standard curve was created by reducing the data using a computer software capable of generating a four parameter logistic (4-PL) curve-fit. As an alternative, a standard curve can be constructed by plotting the mean absorbance for each standard on the x-axis against the concentration on the y-axis and draw a best fit curve through the points on the diagram. The data may be linearized by plotting the log of the GPX_1 concentrations versus the log of the O.D. and the best fit line can be determined by regression analysis.

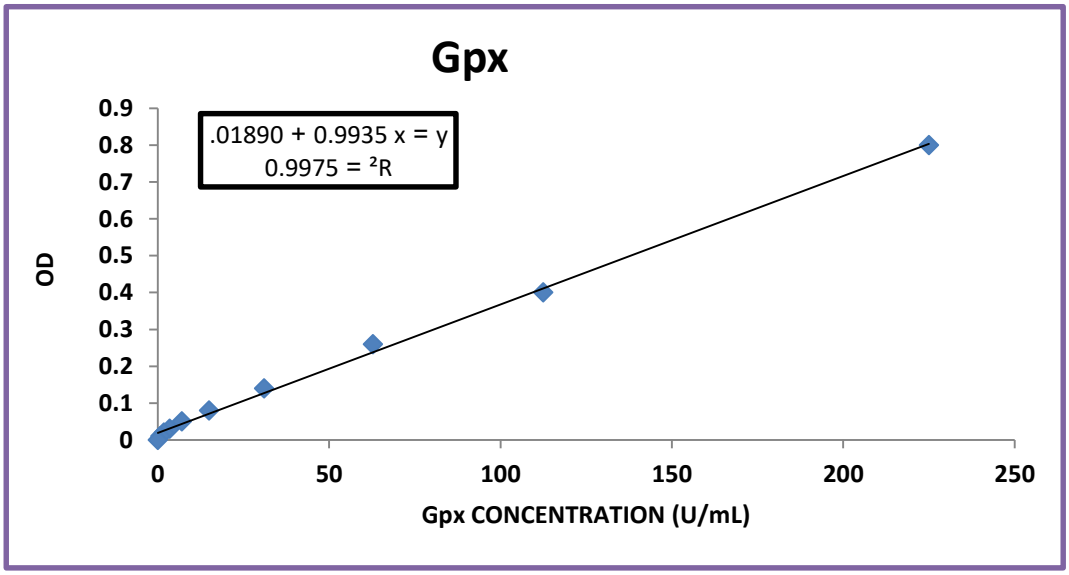

Figure: Standard curve for GPX_1 determination

3-Measurement of serum Superoxide dismutase concentration (SOD).

Test principle

This ELISA kit uses Competitive-ELISA as the method. The microtiter plate provided in this kit has been pre-coated with SOD1. During the reaction, SOD1 in the sample or standard competes with a fixed amount of SOD1 on the solid phase supporter for sites on the Biotinylated Detection Ab specific to SOD1. Excess conjugate and

unbound sample or standard are washed from the plate, and Avidin conjugated to Horseradish Peroxidase (HRP) is added to each microplate well and incubated. Then a Tetramethylbenzidine (TMB) substrate solution is added to each well. The enzyme-substrate reaction is terminated by the addition of a sulphuric acid solution and the color change is measured spectrophotometrically at a wavelength of 450 nm ± 2 nm. The concentration of SOD1 in the samples is then determined by comparing the OD of the samples to the standard curve.

Assay procedure

Bring all reagents and samples to room temperature before use.Centrifuge the sample again after thawing before the assay. All the reagents should be mixed thoroughly by gently swirling before pipetting. Avoid foaming. It's recommended that all samples and standards be assayed in duplicate.

Appendix: 6

lipid profile test

Estimation of serum cholesterol concentration:

Principle:

Easter of cholesterol+H2O $Chol.\ estease$ Cholesterol + Fatty acids Cholesterol +O2 $Chol.\ oxidase$ Cholest-4-en-one+H2O2

H2O+4-Aminophenazone + phenol $peroxidase$ Quinonimine

Reagent:

Reagent (1) Buffer solution: pipes PH 6.9 mmol/L, phenol 26 mmol/L

Reagent (2) vial of enzyme: cholesterol oxidase 300 U/L, peroxidase 1250 U/L, cholesterol esterase 300 U/L, 4-aminophenazone 0.4 mmol/L

Reagent (3): cholesterol standard 200 mg/dl

1. Manual procedure: Cholesterol concentration in serum samples was measured according to the following

a. Reagent and serum samples were brought to room temperature

b. Serum sample, blank and standard were treated as follow:

c. Tube contents were mixed and left to stand for 5 minutes at 37°C before reading

d. the absorbance of the standard was measured and sample was read via spectrophotometer at wavelength 505 nm against the blank

Tubes	Blank	Standard	Sample
Cholesterol standard (s)	-	10 ml	-
Sample	-	-	10 ml
Working Reagent	1ml	1ml	1ml

Calculation:

Appendices

Result were calculated according to the following equation:Total Cholesterol concentration = (O.D sample)/ (O.D/ standard)× nn = 200 mg/dl

Estimation of serum triglyceride concentration (mg/dl):

Principle:

Triglyceride $lipoprotein\ lipase$ Glycerol + fatty acid

Glycerol + ATP $Glycerol\ kinase$,++ Glycerol-3-phosphate+ADP

Glycerol-3-P+O2 $3{-}G{-}P{-}oxidase$ Dihydroxyacetone ne-p+H2O

H2O2+4-Aminophenazone+p+Chlorophenol$peroxidase$ Quinonimine+ H2O

Reagent:

Reagent (1) buffer solution: pipes buffer PH 7.2, 50 mm0l/L, p- chlorophenol 2 mmol/L

Reagent (2) Enzyme: lipoprotein lipase 150 000 U/I, glycerol kinase 800 U/U/I, glycerol-3-phosphate oxidase 4000 U/I, peroxidase 440 U/I, 4-aminophenazone 0.7 mmol/L, ATP 0.3 mmol/L.

Reagent (3) triglyceride Standard (S): Glycerol 200mg/dl.

Procedure:

Triglyceride concentration in serum samples was measured according to the

following:

a. Wave length/filter. 505nm (Hg546nm)/green

b. Temperature 37°C/R. T

c. Light path 1 cm

Pipette into clean dry test tubes labelled as Blank (B), standard (S), and Test(T). Mix well and incubated at 37° C for 5 min or at R. T (25° C) for 15min. measure the absorbance of the standard

Calculation:

Results were calculated according to the following equation:

Triglyceride concentration mg/dl = (O.D sample) / (O.D standard) × n = 200 mg/dl

Appendices

Additive sequence	Blank	Standard	Test
Working reagent	1.0	1.0	1.0
Distilled water	0.01	-	-
Triglyceride	-	0.01	-
standard			
Sample	-	-	0.01

Estimation of serum HDL-Cholesterol concentration (mg/dl):

Principle:

Cholesterol esters + H2O $Chol.esterase$ Cholesterol + fatty acid

Cholesterol +½O2 + H2O $Chol.oxidase$ Cholestenone + H2O2

2 H2O2 + 4-Aminoantipyrine + DCFS $peroxidase$ Quinoneimine + 4H2O

Reagent

Reagent (1) Good's buffer (pH 6.6)100 mmol/l, cholesterol esterase 1400 U/l, cholesterol oxidase 800 U/l, catalase 600 kU/l, N-(2-hydroxy-3-sulfopropyl)-3,5-dimethoxyaniline (HDAOS) 0.6 mmol/l

Reagent (2) Good's buffer (pH 7.0) 100 mmol/l peroxidase 3 kU/l 4–aminoantipyrine (4-AA) 4 mmol/L

Tubes	Blank	Standard	Sample
Distilled water	50 ml	-	-
Cholesterol standard (S)	-	50 ml	-
Sample supernatant	-	-	50 ml
Reagent	1.0 ml	0 ml	1 ml

Appendices

Procedure:

HDL-Cholesterol concentration in serum sample was measured according to the following steps: serum sample 40 – 60 mg/dl 1.04 1.55mmol/l, wavelength 600 nm, temperature 37°C CORMAY HDL DIRECT is intended for automated analysers.

a. Reagent (A, B) and serum sample were brought to room temperature.

b. Serum sample, blank and standard were treated as followed:

c. 0.2 ml of sample was mixed with 0.5 ml of reagent (A) in centrifuge tube and let stand for 10 minute at room temperature.

d. Centrifuged at a minimum of 4000 r.p.m. for 10 minutes.

e. The temperature was collected carefully.

f. Sample supernatant, blank, standard and reagent (B)were treated as follows:

g. Tubes contents were mixed thoroughly and incubated for 10 minute at 37°C.

h. the absorbance (A) of the standard was measured and sample was read via spectrophotometer at wave length 500 nm against the blank.

Calculation: results were calculated according to the following equation:

HDL-cholesterol concentration in the sample (mg/dl) = (Absorbance of the sample/Absorbance of standard) × concentration of standard × sample dilution factor (1.7).

Estimation of serum LDL-Cholesterol concentration (mg/dl):

Principle:

Cholesterol ester $chol.esterase$ chol. + fatty acid

Cholesterol + O2 $Chol.oxidase$ chol. H2O2

2H2O2 $catalase$ H2O + O2

Reagent:

Reagent (1) Good's buffer (pH 7,0) 50 mmol/l, cholesterol esterase 600 U/l, cholesterol oxidase 500 U/l, catalase 1200 kU/l, ascorbate oxidase 3 kU/l, TOOS [N-Ethyl-N-(2-hydroxy-3-sulfopropyl)-3-methylaniline] 2.0 mmol/l

Reagent (2) Good's buffer (pH 7,0) 50 mmol/l, peroxidase 5 kU/l, 4–aminoantipyrine (4-AA) 4 mmol/l.

Appendices

Procedure:

wavelength 600 nm, temperature 37°C, CORMAY LDL DIRECT is intended for automated analysers. serum/plasma < 100 mg/dl < 2.59 mmol/l.

As LDL cholesterol is affected by a number of factors such as smoking, exercise, hormones, age and sex, each laboratory should

establish its own reference ranges for local population.

الخلاصه

أجريت هذه الدراسة في كلية الطب البيطري / جامعة كربلاء لبحث تأثير مسحوق الطماطم في تقليل الضرر الناتج عن تعرض دجاج اللاحم للأفلاتوكسين عن طريق قياس بعض المعايير الكيميوحيوية ومضادات الأكسدة والمناعة والأداء الانتاجي والتغيرات النسيجية للكبد.

استمرت التجربة لمدة 35 يومًا من 12 فبراير إلى 19 مارس/ 2022. وزعت مئة فرخ من افراخ اللحم (روز 308) في عمر يوم واحد بشكل عشوائي إلى 5 مجموعات متساوية (20 لكل مجموعة). المجموعة الأولى اعتبرت مجموعة سيطرة حيث غذيت على عليقة اساسية خالية من اي اضافات خلال مدة التجربة، المجموعة الثانية غذيت يوميا لغاية عمر 28 يوم على عليقة ملوثة بسموم الأفلاتوكسين بنسبة 30 جزء لكل بليون فقط، المجموعة الثالثة غذيت على العليقة الملوثة بسموم الأفلاتوكسين 30 جزء في البليون مكمل بتركيز ميكوفكس 0.1 مجم / كجم علف، المجموعة الرابعة غذيت بعليقة ملوثة بالأفلاتوكسين 30 جزء في البليون ومدعم بمسحوق الطماطم بتركيز 10 جم / كجم علف، بينما غذيت المجموعة الخامسة على عليقة غذائية مدعمة بمسحوق الطماطم بتركيز 10 جم / كجم علف خلال مدة التجربة. تم تحليل مكونات مسحوق الطماطم بواسطة تقنية HPLC.

في اليوم الرابع ، الثامن والعشرين ، الخامس والثلاثين من العمر ، تم جمع عينات دم من 5 دجاجات لكل مجموعة من الوريد الوداجي في أنبوب اختبار بدون مضاد للتخثر ، للحصول على المصل لتقيم الفحوصات الكيميوحيوية والمناعية.

أظهرت نتائج الصفات الكيميوحيوية للدراسة الحالية أن المجموعة AF &TP أظهرت انخفاضًا معنويًا ($P<0.05$) في تركيز AST و ALT ، وكذلك مستوى الكرياتينين مقارنة بمجموعة AF ، كما أظهرت زيادة معنوية ($P<0.05$) في تركيز نفس الصفات في اليوم 28، اما في اليوم 35 أظهرت انخفاض معنوي ($P<0.05$) في تركيز مستوى الكرياتينين مقارنة مع مجموعة AF، بينما انخفضت مستويات الكوليسترول ،الدهون الثلاثية، LDL، VLDL بشكل معنوي ($P <0.05$) في مجموعة AF &TP مقارنة بمجموعة AF التي أظهرت زيادة معنوية ($P<0.05$)، كما زاد معدل HDL معنويا ($P <0.05$) في مجموعة AF &TP مقارنة بمجموعة AF التي أظهرت انخفاضا معنويا ($P<0.05$) في اليومين 28 و 35، كانت الزيادة معنوية ($P<0.05$) في معدلات Gpx و SOD في مجموعة AF &TP مقارنة بمجموعة AF كما انخفضت النتائج معنوياً ($P<0.05$) في تركيز MDA في مجموعة AF &TP مقارنة بمجموعة AF في اليوم 28، اما في اليوم 35 أظهرت زيادة معنوية ($P<0.05$) في تركيز Gpx في مجموعة AF &TP مقارنة بمجموعة AF.

أظهرت نتائج الصفات الحسية زيادة معنوية ($P<0.05$) في الطراوة والعصارة والنكهة واللون والاستساغة في مجموعة AF &TP مقارنة بمجموعة AF كما وأظهرت النتائج وجود زيادة معنوية ($P<0.05$) في وزن الطحال والغدة الزعترية والجراب فابريشيا في مجموعة AF &TP

مقارنة بمجموعة AF بالإضافة إلى انخفاض معنوي (P<0.05) في معدل وزن الكبد في مجموعة AF &TP مقارنة بمجموعة AF في اليوم 28، بينما في اليوم 35 أظهر انخفاضًا معنويًا (P<0.05) في وزن الكبد في مجموعة AF &TP مقارنة بمجموعة AF. أظهرت نتيجة المؤشر الكبدي الجسدي انخفاضاً معنوياً (P<0.05) في مجموعة AF &TP مقارنة بمجموعة AF. أظهرت النتائج المناعية زيادة معنوية (P<0.05) في مستويات الأجسام المضادة لمرض نيوكاسل ومرض الجراب المعدي لمجموعة AF &TP مقارنة بمجموعة (AF)، والتي أظهرت انخفاضًا معنويًا (P<0.05) في تلك الصفات. أظهرت نتائج وزن الجسم في اليوم 7،14،21 و 28 زيادة معنوية (P≤0.05) في مجموعة AF &TP مقارنة بمجموعة AF لنفس النتائج.

أظهرت التغيرات النسيجية المرضية للكبد تحسناً في الأنسجة التالفة لمجموعة AF &TP مقارنة بمجموعة AF عن طريق تقليل النزف وعودة الأنسجة نسبيا إلى التركيب الطبيعي. نستنتج من هذه الدراسة أن تركيز مسحوق الطماطم 10 جم / كجم له دور وقائي لتقليل الضرر الناتج عن التعرض لـ 30 جزء في البليون من الأفلاتوكسين في العلف في فروج اللحم.

جمهورية العراق

وزارة التعليم العالي والبحث العلمي

جامعة كربلاء

كلية الطب البيطري

تأثير مسحوق الطماطمة على الاداء الانتاجي والحالة الصحية في عليقة الدجاج اللاحم المعرض للأفلاتوكسين

رسالة

مقدمة إلى مجلس كلية الطب البيطري في جامعة كربلاء كجزء من متطلبات نيل درجة الماجستير في فرع الصحة العامة

كتبت بواسطة

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