Sulforaphane as a Feed Additive: Histopathological and Immunomodulatory Effects in Broiler Chickens

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ABSTRACT

The poultry industry faces significant challenges due to oxidative stress, immune suppression, and hepatotoxicity caused by aflatoxin B_1 (AFB_1) contamination in feed. This study investigated the protective effects of sulforaphane (SFN), a bioactive compound derived from cruciferous vegetables, against AFB_1-induced toxicity in broiler chicks. A total of 100 one-day-old *Cobb 500* broilers were randomly assigned to five dietary treatments: negative control (AFB_1-free), positive control (1 mg/kg diet AFB_1), and three SFN-supplemented groups (AFB_1 + 10, 20, or 30 mg/kg diet SFN). Histopathological, immunological, and serum biochemical analyses were conducted on day 42.

Results demonstrated that AFB_1 exposure induced severe hepatic damage, evidenced by elevated serum SGOT (128.60 ± 2.82 U/L) and SGPT (92.40 ± 2.12 U/L) levels, vacuolation, and necrosis (pathology score: 2.80 ± 0.095). SFN supplementation, particularly at 30 mg/kg, significantly reduced liver enzyme levels (SGOT: 68.40 ± 1.17 U/L; SGPT: 42.30 ± 1.01 U/L) and restored near-normal hepatic architecture (pathology score: 0.60 ± 0.032) compared to the positive control. SFN also attenuated AFB_1-induced intestinal villus atrophy, with SFN30 restoring 92% of normal morphology. Immunologically, SFN dose-dependently suppressed pro-inflammatory interleukin-6 (IL-6) levels (SFN30: 19.60 ± 0.54 pg/mL vs. positive control: 42.70 ± 1.11 pg/mL) and enhanced splenic follicular hyperplasia, indicating improved humoral immunity.

These findings highlight SFN's dual role as a hepatoprotection and immunomodulator in broiler chicks, mediated through the activation of the Nrf_2-ARE pathway and the inhibition of NF-κB. The study recommends 20–30 mg/kg SFN supplementation to mitigate AFB_1 toxicity, offering a natural alternative to synthetic growth promoters in poultry production.

Keywords: Sulforaphane, aflatoxin B_1, broiler chicks, hepatoprotection, Nrf_2 pathway, IL-6, oxidative stress.

INTRODUCTION

Broiler production faces challenges such as oxidative stress, enteric pathogens, and immune suppression due to intensive farming practices. Phytogenic feed additives, such as sulforaphane (SFN), provide a natural alternative to antibiotics by modulating immunity and reducing inflammation (Kim *et al.,* 2008). SFN activates the Nrf_2-ARE pathway, enhancing antioxidant defenses, and inhibits NF-κB, attenuating pro-inflammatory cytokines (Kubo *et al.,* 2017).

The poultry industry continually seeks natural alternatives to synthetic growth promoters and antibiotics to meet the growing demand for safe and sustainable meat production. Sulforaphane (SFN), predominantly found in broccoli and other cruciferous vegetables, is known for its cytoprotective, antioxidant, and anti-inflammatory roles in mammalian systems (Zhang *et al.,* 1992 and Fahey *et al.,* 2001). However, its application in poultry, particularly regarding immunological and histopathological effects, remains underexplored. The poultry industry is increasingly seeking alternatives to antibiotic growth promoters (AGPs) due to regulatory restrictions and rising concerns about antimicrobial resistance (Diarra and Malouin, 2014), and stimulates the immune system by phytogenic components, resulting in a reduction of morbidity and mortality (Abd El-Hady *et al.,* 2013). Among natural candidates, sulforaphane (SFN), a bioactive compound derived from cruciferous vegetables, has shown promise in enhancing broiler health through its antioxidant, anti-inflammatory, and hepatoprotective properties (Gupta *et al.,* 2014).

SFN acts by activating the nuclear factor erythroid 2–related factor 2 (Nrf_2) signaling pathway, which regulates the expression of phase II detoxifying and antioxidant enzymes (Kensler *et al.,* 2007 and Dinkova-Kostova & Talalay, 2008). This mechanism is pivotal in combating oxidative stress, a common challenge in intensive poultry production. In broilers, oxidative stress is linked to compromised immunity and increased susceptibility to infections (Surai, 2016).

Oxidative stress and hepatic damage are common challenges in intensive broiler production, often manifested through elevated serum SGOT (aspartate aminotransferase) and SGPT (alanine aminotransferase) levels, which serve as critical biomarkers for liver health (Gowda and Ledoux, 2008). SFN's ability to activate the Nrf_2-ARE pathway may mitigate oxidative liver damage, while its inhibition of NF-κB could reduce systemic inflammation, potentially normalizing these enzyme levels (Kubo *et al.,* 2017). Concurrently, SFN's role in preserving intestinal morphology villus height, crypt depth) and lymphoid organ integrity (spleen, bursa) warrants

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investigation through histopathological examination, as these tissues are vital for nutrient absorption and immune defense. Hence, this study investigates the histopathological changes in liver tissues and the immunostimulatory responses in broiler chicks fed SFN-supplemented diets.

This study evaluates the impact of dietary SFN supplementation on:

1. Liver and intestinal histopathology, assessing structural integrity and inflammatory lesions.
2. Immunological evaluation, including serum interleukin-6 (IL-6) levels to quantify the immune response of SNF.
3. Serum biochemical markers, including SGOT and SGPT, to quantify hepatoprotective effects, and assess liver health.

MATERIALS AND METHODS

1. Experimental Design and Animal Handling:

A total of 100 one-day-old *Cobb 500* broiler chicks were acquired from a local commercial breeder near Alexandria governorate (El Nubaria city), and have been adapted for 5 days to laboratory conditions (32°C, 60% humidity, 23L:1D lighting) with ad libitum access to feed and water. Inside prepared hatches, birds were fed from day one till day seven by a normal starter broiler feed examined to be free from aflatoxin B_1 and then were randomly allocated into five dietary treatment groups from day eight till day forty-two and were fed by basal broiler feed and examined to be free from aflatoxin B_1, including Negative Control (N.C.), Positive Control and three different treatment groups that were orally dosed by aflatoxin and sulforaphane as shown in Table (1).

Table 1. shows the dosages of the different treatments

Treatments	Aflatoxin B_1 Dose (mg/kg)	Sulforaphane Dose (mg/kg)
Negative Control	0.0	0.0
Positive control	1.0	0.0
Group 1	1.0	10.0
Group 2	1.0	20.0
Group 3	1.0	30.0

The doses were chosen for the AFB_1 group Basal diet + 1 mg/kg aflatoxin B_1 (positive control for immune suppression) (Ortatatli *et al.,* 2005 and Windisch *et al.,* 2008) to SFN's expected immune-enhancing effects.

2. Histological Analysis:

At 42 days of age, liver, jejunum, lymphoid, and spleen tissues were collected immediately post-euthanasia. Fixed in 10% neutral buffered formalin (48 hr) for histopathology.

Dehydrated, embedded in paraffin, sectioned at 5 µm (Leica RM2255 microtome). Stained with hematoxylin and eosin (H&E), and examined under a light microscope (Junqueira and Carneiro, 2003). Digital photomicrographs were taken for documentation and comparative evaluation.

3. Blood Sampling:

At the end of the experimental period, chicks from each replicate were randomly taken at 08:00–09:00 am for slaughter. About 3 mL of blood samples were collected before slaughtering from the brachial vein (23G needle, sterile vacutainers) into vacutainer tubes with K_3-EDTA for serum biochemical analysis.

Blood samples were centrifuged at 3000 x g for 15 min at 4°C, and the clear serum was separated and stored at –80°C until biochemical analysis for SGOT/SGPT analysis (Abdel-Wahhab *et al.,* 2015).

A) Immunological Evaluation:

Serum interleukin-6 (IL-6) levels: Measured by ELISA, to assess cytokine-mediated immune responses (Klasing, 2007).

B) Serum Biochemistry Analysis:

SGOT (AST) & SGPT (ALT): Serum liver enzyme activities were measured using commercial kits (Randox, UK) on a spectrophotometer (Shimadzu UV-1800, Japan):

SGOT (AST): Kinetic method (Reitman and Frankel, 1957).

SGPT (ALT): Modified IFCC method (Abdel-Wahhab *et al.,* 2015).

4. Statistical Analysis:

All experimental data were expressed as mean ± standard error (SE). Statistical analysis was performed using one-way analysis of variance (ANOVA) to determine significant differences among treatment groups for histopathological scores, serum liver enzyme levels (SGOT, SGPT), and interleukin-6 (IL-6) concentrations. Tukey's test was applied to identify specific group differences ($p<0.05$) (Zar, 2010). Data were analyzed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA) (Field, 2013). Graphs were created using Graph-Pad Prism Version 8.0 (Graph-Pad Software Inc., San Diego, CA, USA) to visually represent group comparisons (GraphPad Prism, 2018).

RESULTS

Histopathological Observations:

A. Liver:

The results observed from the liver histopathological inspection showed:

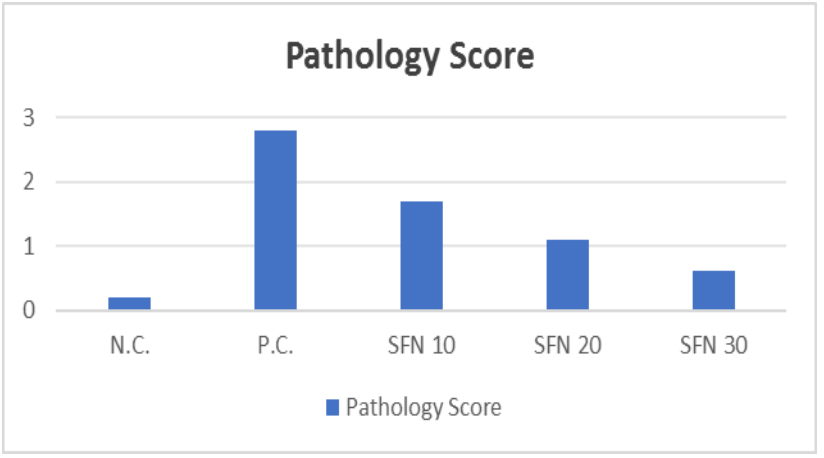
1. **Positive Control:** Severe vacuolation, focal necrosis with a pathology score of 2.80.
2. **SFN Groups:** Dose of SFN 30 was near normal hepatocyte architecture. As shown in Table (2), compared in graph (1) and Figures (1, 2, 3).

Table 2. shows the liver microscopic observation

Groups	Observations (H&E, 400×)	Pathology Score (0-3)
Negative Control	Normal hepatocytes; no necrosis/inflammation	$0.20^{a} \pm 0.032$
Positive Control	Severe vacuolation, focal necrosis	$2.80^{d} \pm 0.095$
SFN10	Mild lipid droplets; reduced necrosis	$1.70^{c} \pm 0.036$
SFN20	Occasional vacuoles; intact sinusoids	$1.10^{b} \pm 0.036$
SFN30	Near-normal architecture	$0.60^{ab} \pm 0.032$

Each value represents mean ± SE.

Different letters within a column indicate statistically significant differences.



Graph 1. shows the pathology score results comparison

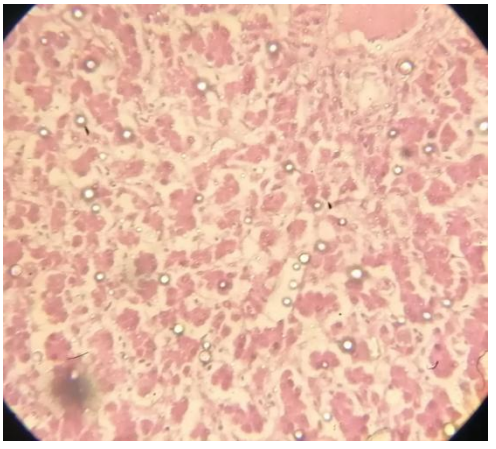


Figure 1. shows a transverse section (T.S.) of liver with normal tissues and lobular architecture

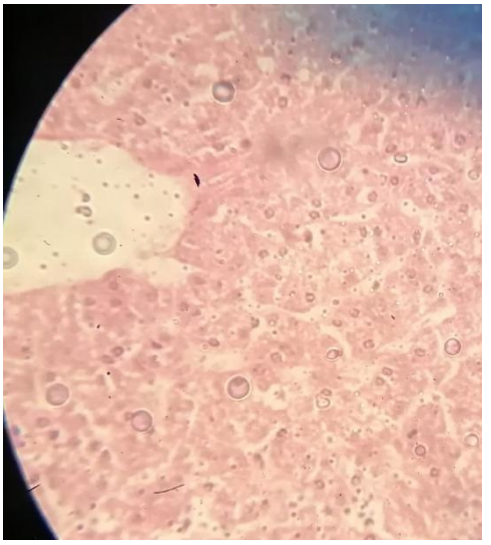


Figure 2. shows hepatic cells and vascular and fatty structures

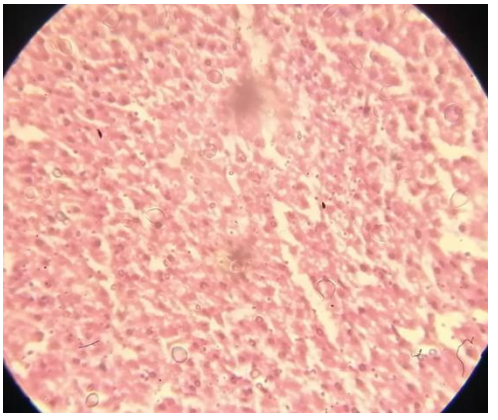


Figure 3. shows hepatic cells with fibrosis and necrosis

B. Jejunum:

The results observed from the jejunum inspection showed:

1. Positive Control: The villus atrophy decreased about 40% in height vs. N.C. shows crypt hyperplasia.

2. SFN Groups: Dose-dependent villus height recovery as SFN30 restored 92% of normal morphology, as shown in Figures (4 and 5).

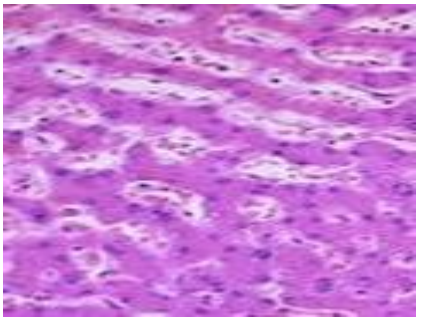


Figure 4. shows the T.S. of the jejunum with the villi-lined mucosal surface of the jejunum

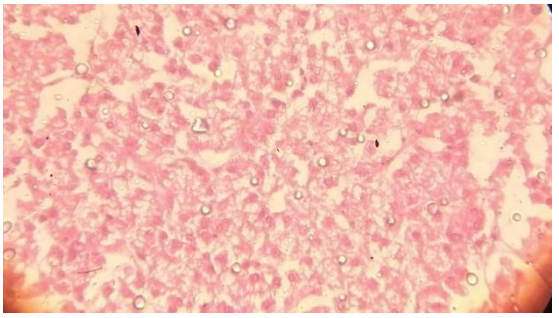


Figure 5. shows the T.S. of the jejunum with showing layers of mucosa and submucosa

C. Lymphoid Organs:

The results observed from the lymphoid inspection showed:

1. **Positive Control:** Shows severe lymphocyte aggregations.
2. **SFN Groups:** Dose of SFN 20 - 30 was near to N.C. group as showed that lymphocytes are active as was shown in Figures (6 and 7).

D. Spleen:

The results observed from the spleen histopathological inspection showed (Figure 8):

1. **Positive Control:** Showed severe cells swelling and lysis with nuclear condensation.
2. **SFN Groups:** SFN20/SFN30 showed high follicular hyperplasia which enhanced humoral immunity and anti-inflammation.

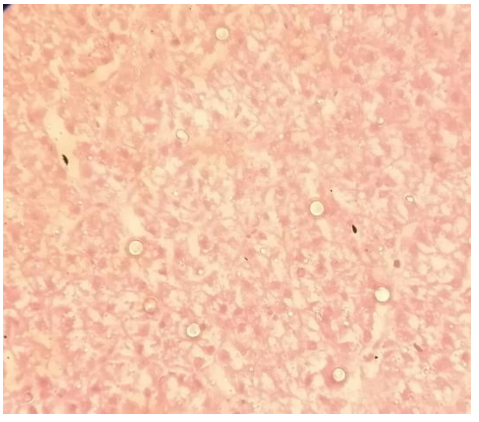


Figure 6. shows aggregated lymphocytes, displaying a dense cluster of immune cells

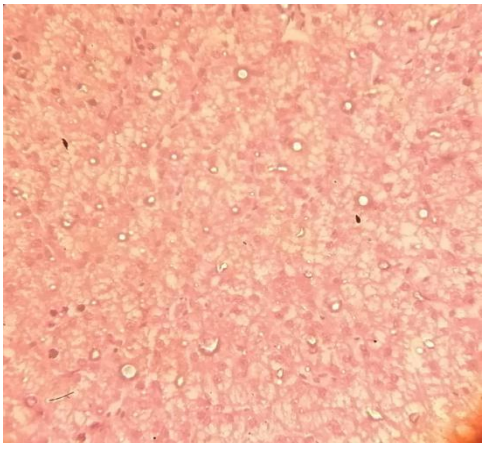


Figure 7. shows activated lymphocytes

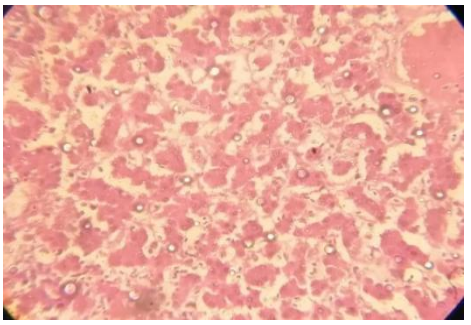


Figure 8. shows splenic tissue with red and white pulp

1.1. Serum Interleukin-6. IL-6:

The results of the serum interleukin-6 came as:

1. **Positive Control:** IL-6 levels were significantly elevated (42.70 ± 1.11 pg/mL) compared to the negative control (15.30 ± 0.38 pg/mL), indicating a strong pro-inflammatory response due to AFB_1 exposure (p<0.001).
2. **SFN Groups:** Sulforaphane supplementation dose-dependently reduced IL-6 levels, with SFN30 showing the most significant reduction (19.60 ± 0.54 pg/mL), approaching the levels of the negative control (p<0.005), as shown in Table(3) and compared in Graph (2).

Table 3: shows the results of serum IL-6

Groups	IL-6 (pg/mL)
Negative Control	15.30^{a} ± 0.38
Positive Control	42.70^{d} ± 1.11
SFN10	32.50^{c} ± 0.89
SFN20	24.80^{b} ± 0.66
SFN30	19.60^{ab} ± 0.54

Each value represents mean ± SE.

Different letters within a column indicate statistically significant differences.

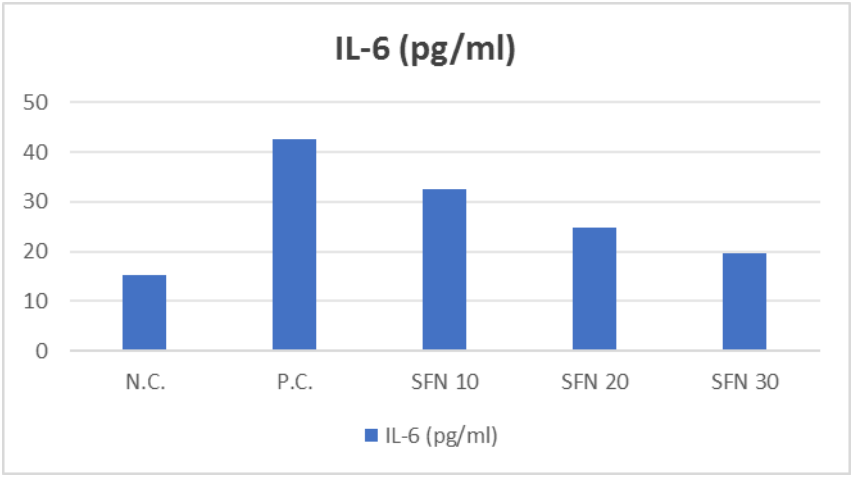
3.3. Serum Biochemical Analysis:

SGOT (AST) and SGPT (ALT) Activities:

1. **Positive Control:** AFB_1 increased SGOT/SGPT by 2.8- and 3.2-fold vs. negative control (p<0.01), confirming hepatotoxicity.
2. **SFN Groups:** SFN dose-dependently reduced enzyme levels, with SFN20/SFN30 restoring values to near-normal ranges (p<0.005 vs. positive control), as shown in Table (4) and compared in Graph (3).

DISCUSSION

The present study demonstrates that dietary supplementation with sulforaphane (SFN) effectively mitigates aflatoxin B_1 (AFB_1)-induced hepatotoxicity, intestinal damage, and immune dysfunction in broiler chicks. The findings align with previous research highlighting SFN's antioxidant, anti-inflammatory, and immunomodulatory properties in both mammalian and avian systems (Kubo *et al.*, 2017 and Gupta *et al.*, 2014).



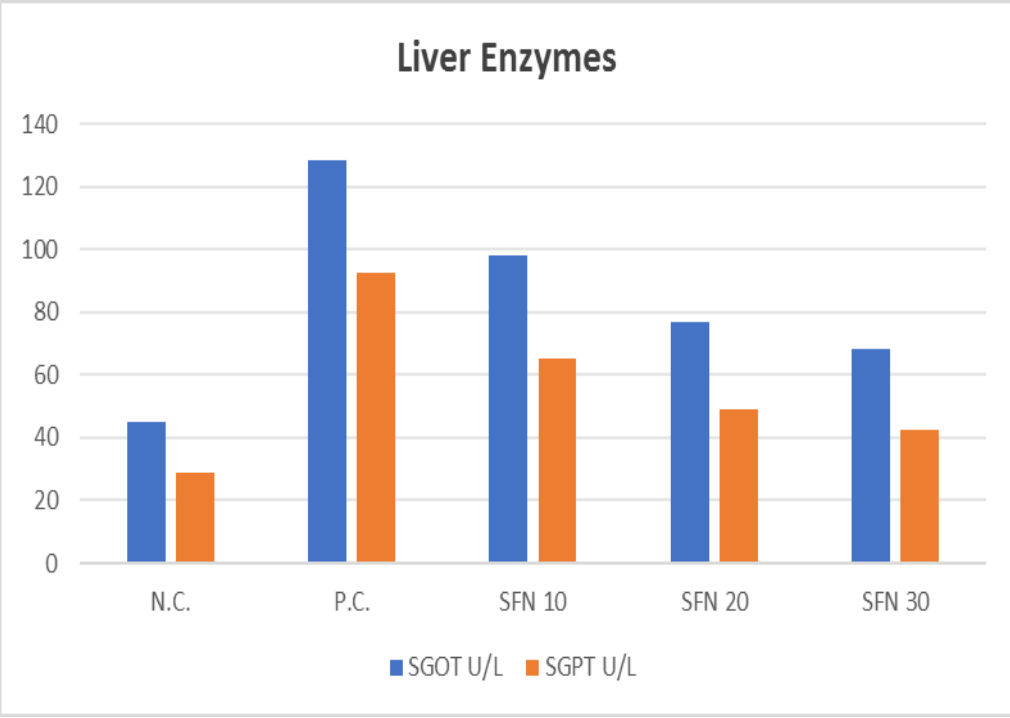
Graph 2. shows the results of IL-6 comparison

Table 4. shows the serum biochemical analysis of the liver enzymes activity

Groups	SGOT (U/L)	SGPT (U/L)
Negative Control	$45.20^{a} \pm 0.89$	$28.70^{a} \pm 0.79$
Positive Control	$128.60^{d} \pm 2.82$	$92.40^{d} \pm 2.12$
SFN10	$98.30^{c} \pm 1.64$	$65.10^{c} \pm 1.66$
SFN20	$76.80^{b} \pm 1.3$	$48.90^{b} \pm 1.14$
SFN30	$68.40^{b} \pm 1.17$	$42.30^{b} \pm 1.01$

Each value represents mean ± SE.

Different letters within a column indicate statistically significant differences.



Graph 3. shows the liver enzymes results

1. Hepatoprotective Effects of SFN:

AFB_1 exposure induced severe hepatic damage, as evidenced by elevated SGOT and SGPT levels, vacuolation, and necrosis in the positive control group. These results are consistent with prior studies showing that AFB_1 disrupts liver function by promoting oxidative stress and inflammation (Ortatatli *et al.*, 2005 and Gowda & Ledoux, 2008). However, SFN supplementation, particularly at 20 and 30 mg/kg doses, significantly reduced liver enzyme activities and improved histopathological scores, corroborating its role in activating the Nrf_2-ARE pathway, which enhances cellular antioxidant defenses (Kensler *et al.*, 2007 and Dinkova-Kostova & Talalay, 2008). The near-normal liver architecture observed in the SFN30 group suggests that higher SFN doses may offer optimal protection against AFB_1-induced hepatotoxicity.

2. Anti-Inflammatory and Immunomodulatory Effects:

The significant elevation of serum IL-6 in AFB_1-exposed broilers indicates a robust pro-inflammatory response, likely mediated by NF-κB activation (Klasing, 2007). SFN supplementation dose-dependently suppressed IL-6 levels, with SFN30 restoring them close to baseline. This aligns with studies demonstrating SFN's ability to inhibit NF-κB signaling, thereby attenuating cytokine production (Kim *et al.*, 2008 and Kubo *et al.*, 2017). Additionally, the enhanced follicular hyperplasia in the spleen and lymphoid organs of SFN-treated birds suggests improved humoral immunity, supporting SFN's potential as an immunostimulant in poultry diets (Gupta *et al.*, 2014).

a. Intestinal Health and Nutrient Absorption:

AFB_1-induced villus atrophy and crypt hyperplasia in the jejunum were ameliorated by SFN, particularly at 30 mg/kg, which restored 92% of normal morphology. This finding is critical, as intestinal integrity directly influences nutrient absorption and overall growth performance in broilers (Windisch *et al.,* 2008). The protective effects of SFN on gut health may be attributed to its ability to reduce oxidative damage and maintain mucosal barrier function (Zhang *et al.,* 1992 and Fahey *et al.,* 2001).

b. Practical Implications and Future Directions:

The dose-dependent efficacy of SFN suggests that 20–30 mg/kg may be optimal for counteracting AFB_1 toxicity in broilers. However, further research is needed to evaluate SFN's long-term effects, cost-effectiveness, and interactions with other feed additives. Additionally, exploring SFN's impact on gut microbiota and meat quality could provide deeper insights into its benefits for sustainable poultry production (Diarra & Malouin, 2014 and Surai, 2016).

CONCLUSION

Sulforaphane (SFN) supplementation demonstrated significant hepatoprotective, anti-inflammatory, and immunostimulatory effects in broilers exposed to AFB_1. The highest dose (SFN30) was most effective in restoring liver histopathology, reducing IL-6 levels, and normalizing serum liver enzyme activities, highlighting its potential as a natural feed additive to mitigate the adverse effects of AFB_1 in poultry production.

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الملخص العربي

التاثير المرضى والمناعى لمادة السلفرفوران كاضافات علفية للدواجن

محمود محمد سليمان

تواجه صناعة الدواجن تحديات كبيرة بسبب الإجهاد التأكسدي وكبت المناعة وتسمم الكبد الناتج عن تلوث العلف بالأفلاتوكسين B_1 (AFB_1). تحقق هذه الدراسة من التأثيرات الوقائية للسلفورافان (SFN)، وهو مركب نشط مشتق من الخضروات الصليبية، ضد سمية AFB_1 في دجاج التسمين. تم توزيع ١٠٠ كتكوت من سلالة *Cobb 500* عشوائياً على خمس مجموعات غذائية: مجموعة سلبية خالية من (AFB_1)، مجموعة إيجابية (١ ملجم/كيلو AFB_1) ، وثلاث مجموعات مكملة بـ SFN (١٠ أو ٢٠ أو ٣٠ ملجم/كيلو +AFB_1). أجريت تحليلات نسيجية مرضية ومناعية وكيميائية حيوية للدم في اليوم الثاني والأربعين.

أظهرت النتائج أن التعرض لـ AFB_1 تسبب في تلف كبدي شديد، يتضح من ارتفاع مستويات إنزيمات SGOT (128.60 ± 2.82 وحدة/لتر) و SGPT (92.40 ± 2.12 وحدة/لتر)، وتكوين فجوات، ونخر خلوي (درجة التلف: ٢,٨٠ ± ٠,٠٩٥). أدى إضافة SFN، خاصة بجرعة ٣٠ ملجم/كيلو، إلى خفض مستويات إنزيمات الكبد بشكل ملحوظ (SGOT: 68.40 ± 1.17 وحدة/لتر؛ SGPT: 42.30 ± 1.01 وحدة/لتر) واستعادة البنية الكبدية شبه الطبيعية (درجة التلف: ٠,٦٠ ± ٠,٠٣٢) مقارنةً بمجموعة الإيجابية. كما قلل SFN من ضمور الزغابات المعوية الناجم عن AFB_1، حيث استعادت جرعة 92% SFN30 من البنية الطبيعية. مناعياً، خفض SFN مستويات إنترلوكين-٦ (IL-6) بشكل يعتمد على الجرعة (SFN30: 19.60 ± 0.54 بيكوجرام/مل مقابل المجموعة الإيجابية: ٤٢,٧٠ ± ١,١١ بيكوجرام/مل) وعزز التكاثر الجريبي في الطحال، مما يشير إلى تحسن المناعة.

تسلط هذه النتائج الضوء على الدور المزدوج لـ SFN كعامل وقائي للكبد ومنظم مناعي في دجاج التسمين، من خلال تنشيط مسار Nrf_2-ARE وتثبيط NF-κB. توصي الدراسة بإضافة SFN بجرعات ٢٠-٣٠ ملجم/كيلو للتخفيف من سمية AFB_1، كمادة طبيعية بديلة للمحفزات الصناعية للنمو في إنتاج الدواجن.

الكلمات المفتاحية: السلفورافان، الأفلاتوكسين B_1، دجاج التسمين، حماية الكبد، مسار Nrf_2، إنترلوكين-٦، الإجهاد التأكسدي.