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Evaluation of Ovalbumin-Induced Asthma Protocols in Wistar Rats: A Comparative Study of Systemic Haematological, Oxidative, and Inflammatory Biomarkers

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ABSTRACT

Background: Ovalbumin (OVA)-induced asthma models are widely employed in experimental research; however, the extent to which different sensitisation and challenge protocols influence systemic biomarkers remains unclear. This study investigated the effects of varying OVA induction methods on haematological indices, oxidative stress markers, and inflammatory cytokines in Wistar rats.
Methods: This study involved 42 female Wistar rats weighing 140-180g, which were acclimatized for one week. They were divided into 6 different groups of 7 rats. During the experimental process, all groups (2-6) were sensitized with 1 mg OVA and 20 mg AlOH dissolved in 0.9% saline on days 1, 7, and 14, and then challenged with 1% OVA biweekly for 28 days. Group 1 control; group 2 sensitized on day 1 and 7, group 3 sensitized on day 1, 7, and 14; group 4 sensitized on day 1 and challenged; group 5 sensitized on day 1 and 7 and challenged; group 6 sensitized on day 1, 7, and 14 and challenged. All animals were euthanized at the end of 28 days. Haematological parameters (white blood cell count, leukocyte differentials, platelet indices), oxidative stress markers (malondialdehyde [MDA], catalase, superoxide dismutase [SOD], glutathione peroxidase [GPx], total protein), and cytokines (IL-10, NF-κB, TNF-α) were quantified and compared with control animals.
Results: No statistically significant differences ($p > 0.05$) were observed in haematological parameters, SOD, GPx, total protein, IL-10, NF-κB, or TNF-α across groups (challenged and sensitized) compared to controls. However, rats sensitised on day 1 and challenged showed a significant increase in serum MDA and catalase activity ($p < 0.05$), indicating mild oxidative stress.
Conclusion: Different OVA sensitisation and challenge methods did not significantly alter systemic haematological or cytokine profiles in Wistar rats, although oxidative stress was evident under specific conditions. These findings suggest that commonly used OVA protocols may be insufficient to induce systemic features of asthma, and oxidative stress markers such as MDA and catalase may provide more sensitive indicators of early systemic alterations. Future studies should employ chronic exposure models and broader biomarker panels to enhance translational relevance.

Key words: Ovalbumin-induced asthma; Wistar rats; haematological indices; oxidative stress; cytokines

INTRODUCTION

Asthma is a chronic inflammatory airway disorder characterized by reversible airflow obstruction, airway hyperresponsiveness (AHR), and persistent inflammation. It affects over 300 million people worldwide and significantly contributes to morbidity and mortality (1). The disease involves multiple inflammatory cells, including eosinophils, neutrophils, macrophages, and Th2 lymphocytes, along with mediators like interleukins and cysteinyl leukotrienes. These factors lead to airway remodeling, which includes goblet cell hyperplasia, subepithelial fibrosis, and smooth muscle hypertrophy (2–4). Systemic immune activation is also observed, as evidenced by leukocytosis, eosinophilia, and platelet alterations, which may serve as biomarkers of disease activity (5,6).

Oxidative stress is a key factor in the development of asthma. An imbalance between reactive oxygen species (ROS) and antioxidant defenses encourages inflammation, tissue damage, and decreased lung function (7,8). Patients often exhibit low antioxidant enzyme activity and high levels of oxidative mediators, especially during attacks (8,9). These findings underscore the potential of antioxidant-based treatments, particularly considering the adverse effects associated with long-term corticosteroid use (10).

Animal models are still crucial for understanding asthma mechanisms and testing treatments (11–13). Ovalbumin (OVA)-induced asthma in rodents, especially Wistar rats, is

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commonly used, showing features like high IgE levels, eosinophilic inflammation, airway hyperresponsiveness (AHR), and remodeling (14). Standard methods include sensitization (usually through intraperitoneal injection) followed by airway challenge. However, injection does not replicate natural inhalation exposure, which might change inflammatory responses (15). Additionally, because asthma varies widely, no single allergen model can fully represent the disease, highlighting the importance of improving sensitization and challenge protocols for better translational relevance (16,17). Cytokines play a key role in asthma immunopathology. NF-κB and TNF-α drive inflammation, while IL-10 offers anti-inflammatory regulation (18). Their levels help indicate disease severity and response to treatment.

This study examines the impact of varying OVA induction methods on lung biomarkers (IL-10, NF-κB, TNF-α) and systemic hematological indices (white blood cell count, leukocyte subsets, platelet count) in female Wistar rats.

MATERIALS AND METHODS

Animals and Ethical Approval

Forty-two female Wistar rats (120–180 g) were obtained from the Animal Facility of the Department of Anatomy, University of Benin, Nigeria. Rats were housed under standard laboratory conditions (a 12-hour light:12-hour dark cycle) with *ad libitum* access to food and water. After one week of acclimatization, rats were randomly assigned to six experimental groups (n = 7). All procedures were approved by the College of Medical Sciences Health Research Ethics Committee, University of Benin (Ref: CMS/REC/2024/711).

Experimental Design and Asthma Induction

The total duration of the study was 8 weeks, including acclimatization (1 week), sensitization (2–3 weeks), and challenge (4 weeks). Asthma was induced using a modified ovalbumin (OVA) protocol (19–21). Sensitization was carried out with intraperitoneal injection of 1 mg OVA and 20 mg AIOH dissolved in 0.9% saline. Sensitized animals were then challenged in a transparent chamber (50 × 40 × 30 cm) filled with aerosolized 1% (w/v) OVA in saline, delivered through a compressor nebulizer (CNB 69009, with an aerosol delivery rate of ≥ 0.25 mL/min) for 15 minutes, twice weekly, for 28 days (22). The negative control group received the same volume of saline for both sensitization and challenge. The experimental groups were as follows:

- **Group 1 (Control):** Received saline via intraperitoneal injection and aerosolized saline challenge.
- **Group 2:** Each rat was sensitized with the same dosage of 1 mg OVA and 20 mg aluminum hydroxide (AlOH) in 0.9% saline intraperitoneally (i.p.) on days 1 and 7. Not challenged.
- **Group 3:** Sensitized i.p. on days 1, 7, and 14. Not challenged.
- **Group 4:** Sensitized i.p. on day 1 only. Challenged.
- **Group 5:** Sensitized i.p. on days 1 and 7. Challenged.
- **Group 6:** Sensitized i.p. on days 1, 7, and 14. Challenged.

Challenged groups (4–6) were exposed to an aerosol of 1% (w/v) OVA in saline for 15 minutes, twice a week for 4 weeks, using a compressor nebulizer (aerosol delivery rate: 0.25 ml/min) in a chamber (70 × 30 × 40 cm).

Sample Collection and Processing

Twenty-four hours after the final challenge, rats were anesthetized using chloroform vapor. Blood was collected via cardiac puncture. For serum isolation, blood was incubated at room temperature for 30 minutes and centrifuged at 2500 rpm for 15 minutes. The serum was aliquot was collected and stored at −4°C until analysis.

Biochemical Analyses

- **Inflammatory markers:** Serum levels of IL-10 (Elabscience: E-EL-R0016), IgE (Fortress diagnostics: BXC0751), IL-!B (Elabscience: E-EL-R0012), and NF-κB (Elabscience: E-EL-R0674) were measured using commercial ELISA kits.
- **Oxidative stress markers:**
  - Malondialdehyde (MDA) was assayed via the thiobarbituric acid reactive substances (TBARS) method (23).
  - Catalase (CAT) activity was determined based on hydrogen peroxide decomposition (24).
  - Superoxide dismutase (SOD) activity was assessed using the adrenaline autooxidation method (25).
  - Glutathione peroxidase (GPx) activity was measured according to the pyrogallol oxidation method (26).
- Total protein was measured to normalize enzyme activities. The total protein concentration was determined using the Bradford colorimetric test, which relies on the interaction of Coomassie Brilliant Blue G-250 dye with proteins, resulting in a change in absorbance (27).

Statistical Analysis

GraphPad Prism version 10.2.2 was used for all analyses. Data were expressed as mean ± standard error of the mean (SEM). Differences among groups were analyzed using one-way ANOVA followed by Tukey's *post hoc* test. A p-value < 0.05 was considered statistically significant.

RESULTS

From table 1, no statistically significant differences ($p > 0.05$) were observed in white blood cell count, lymphocyte percentage, mid-sized white blood cells, granulocytes, platelet count, mean platelet volume, or plateletcrit among rat groups sensitized on days 1 and 7 (not challenged), sensitized on days 1, 7, and 14 (not challenged), sensitized on day 1 (challenged), sensitized on days 1 and 7 (challenged), and sensitized on days 1, 7, and 14 (challenged), compared to control.

From table 2, a statistically significant increase ($p < 0.05$) was observed in serum malondialdehyde activity and serum catalase activity in the rat group that was sensitized on day 1 and subsequently challenged. No statistically significant differences ($p > 0.05$) were observed in serum superoxide dismutase activity, serum glutathione peroxidase activity, or serum total protein activity among rat groups sensitized on days 1 and 7 (not challenged), sensitized on days 1, 7, and 14 (not challenged), sensitized on days 1 and 7 (challenged), and sensitized on days 1, 7, and 14 (challenged), compared to control. ($^{*}p<0.05$ compared to control)

Table 3 showed no statistically significant differences ($p > 0.05$) were observed in serum IL-10, NF-κB, or TNF-α concentration among rat groups sensitized on days 1 and 7 (not challenged), sensitized on days 1, 7, and 14 (not challenged), sensitized on day 1 (challenged), sensitized on days 1 and 7 (challenged), and sensitized on days 1, 7, and 14 (challenged), compared to control.

Table 1: Haematological changes in varying ovalbumin asthma induction methods

	Control	Sen 1 & 7	Sen 1, 7 &14	Sen 1/Challenged	Sen 1 & 7/Challenged	Sen 1, 7 & 14/Challenged
WBC ($x10^3/\mu L$)	5.21±0.35	5.13±0.61	6.54±1.48	5.56±0.68	6.412±1.60	5.49±0.73
LYM (%)	86.03±1.24	83.85±1.70	83.45±1.33	89.28±1.05	81.35±2.87	81.49±3.32
MID (%)	10.64±0.81	11.47±1.12	12.33±1.19	8.54±0.93	13.8±1.709	12±0.72
GRAN (%)	3.33±0.47	4.68±0.72	4.223±0.53	2.18±0.12	4.85±1.17	3.66±0.21
PLT ($x10^3/\mu L$)	630.70±32.406	454.20±39.39	577.90±102.40	654.00±35.97	644.20±62.70	630.60±57.09
MPV (fL)	7.443±0.05281	7.533±0.1498	7.213±0.1156	7.44±0.1077	7.4±0.1983	7.27±0.07
PCT (%)	0.46±0.02	0.34±0.034	0.41±0.07	0.48±0.03	0.47±0.05	0.45±0.042

Table 2: Serum oxidative stress and protein activity in varying ovalbumin asthma induction methods

	Control	Sen 1 & 7	Sen 1, 7 &14	Sen 1/Challenged	Sen 1 & 7/Challenged	Sen 1, 7 & 14/Challenged
T. Protein (g/dL)	3.35±0.034	3.60±0.18	3.38±0.05	3.16±0.04	3.37±0.08	3.29±0.02
SOD (U/g protein)	0.55±0.01	0.53±0.03	0.55±0.00	0.60±0.01	0.56±0.01	0.56±0.00
CAT (U/g protein)	0.05±0.00	0.07±0.00	0.08±0.00	0.13±0.02***	0.09±0.01	0.08±0.01
GPx (U/g protein)	0.93±0.03	0.89±0.04	0.89±0.02	1.01±0.02	0.94±0.02	0.93±0.02
MDA (mol/g protein)	0.27±0.00	0.24±0.01	0.30±0.02	0.40±0.01**	0.28±0.01	0.28±0.05

Table 3: Serum cytokine and transcription factor changes in varying ovalbumin asthma induction methods

	Control	Sen 1 & 7	Sen 1, 7 &14	Sen 1/Challenged	Sen 1 & 7/Challenged	Sen 1, 7 & 14/Challenged
IL-1B (pg/ml)	38.78±2.376	53.86±5.33	46.73±4.94	46.98±5.54	47.31±3.348	46.75±5.827
IL-10 (pg/ml)	2.24±0.17	2.07±0.11	3.05±0.3782	2.33±0.13	2.49±0.44	2.61±0.66
IgE (IU/ml)	473.90±6.87	494.20±2.89	463.60±22.39	436.00±11.35	469.50±11.98	483.90±31.86
NF-κB (ng/ml)	133.40±18.45	138.70±13.29	136.20±12.83	123.10±10.94	158.00±17.40	139.50±3.42

DISCUSSION

This study investigated the effects of various ovalbumin (OVA) sensitisation and challenge protocols on haematological indices, oxidative stress/antioxidant enzyme activities, and inflammatory cytokines in Wistar rats. Overall, the findings suggest that these induction methods did not produce substantial systemic alterations in most measured parameters, with only limited evidence of oxidative stress in specific groups.

No statistically significant differences were observed in white blood cell (WBC) count, lymphocyte percentage, mid-sized white blood cells, granulocytes, platelet count, mean platelet volume, or plateletcrit between the experimental groups and controls. These results suggest that variations in sensitisation and challenge protocols did not elicit systemic haematological changes in Wistar rats.

Typically, allergic asthma models are associated with elevated leukocyte and lymphocyte counts due to airway inflammation (7,28). Similarly, granulocyte infiltration, especially eosinophils, is a hallmark of allergic inflammation (29). The absence of such findings here may reflect differences in rat strain, antigen dose, sensitisation route, or duration of exposure. Moreover, platelet parameters remained unchanged, despite prior evidence suggesting that platelet activation contributes to airway hyperresponsiveness (30). These results indicate that acute systemic haematological indices may not accurately reflect the immunological changes induced by OVA in this model.

Among oxidative stress markers, a significant increase in serum malondialdehyde (MDA) activity was observed in the group sensitised on day 1 and challenged, indicating lipid peroxidation and oxidative stress. This was accompanied by a corresponding increase in catalase activity, suggesting an adaptive antioxidant response to elevated reactive oxygen species (ROS). These findings are consistent with earlier studies that have linked OVA-induced immune activation to increased oxidative stress (31,32).

Conversely, no significant changes were observed in serum superoxide dismutase (SOD), glutathione peroxidase (GPx), or total protein levels across groups. This stability aligns with reports that antioxidant responses are context-dependent and may remain unchanged under mild oxidative stress (33,34). The selective rise in MDA and catalase suggests that oxidative stress occurred, but was neither severe nor sustained enough to disrupt systemic redox balance broadly.

No statistically significant differences were found in serum IL-10, NF-κB, or TNF-α concentrations between the experimental groups and the controls. IL-10, an anti-inflammatory cytokine, is typically upregulated following immune activation, while NF-κB and TNF-α are central mediators of inflammatory signalling (35,36). The lack of changes suggests that the OVA protocols used in this study were insufficient to induce systemic inflammatory responses.

Discrepancies with previous studies reporting elevated NF-κB and TNF-α (18) may be attributed to differences in OVA dose, adjuvant concentration, or the timing of sample collection. It is possible that immune activation was localised to the airways without manifesting in measurable systemic cytokine changes.

Taken together, the results suggest that the OVA sensitisation and challenge protocols used here were insufficient to elicit robust systemic haematological, oxidative, or inflammatory alterations. While localised airway inflammation may still have occurred, systemic markers, including WBC indices, cytokines, and most antioxidant enzymes, remained largely unaffected. The only consistent systemic changes were elevated MDA and catalase activity in the day 1 sensitised and challenged group, indicating mild oxidative stress.

These findings highlight important methodological considerations: OVA-induced asthma models are highly dependent on factors such as antigen dose, adjuvant, timing, and species. Haematological indices and serum cytokines may not always provide sensitive readouts of airway inflammation, whereas oxidative stress markers may better capture early systemic effects.

Conclusion: The different OVA sensitisation and challenge methods employed in this study did not significantly alter systemic haematological indices, serum cytokine levels, or most antioxidant enzymes in Wistar rats. Only malondialdehyde and catalase activities were significantly elevated in rats sensitised on day 1 and challenged, indicating mild oxidative stress. These findings suggest that the protocols tested may not be optimal for reliably inducing systemic features of asthma.

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REFERENCES

1. Fahy, J. V. Type 2 inflammation in asthma — present in most, absent in many. National Reviews. *Immunology*. 2015; 15(1), 57.
2. Hammad, H. & Lambrecht, B. N. The basic immunology of asthma. *Cell*. 2021; 184(6), 1469-1485.
3. Martin, J. G., & Tamaoka, M. Rat models of asthma and chronic obstructive lung disease. Vol. 19, Pulmonary Pharmacology and Therapeutics. 2006.
4. Lambrecht, B. N, & Hammad, H. The airway epithelium in asthma. *Nature Medicine*. 2012; 18(5), 684–92.
5. Komlósi, Z. I., van de Veen, W., Kovács, N., Szűcs, G., Sokolowska, M., O'Mahony, L., Akdis, M., & Akdis, C. Cellular and molecular mechanisms of allergic asthma. Molecular Aspects of Medicine. 2022; 85, 100995.
6. Barnes, P. J. Cellular and molecular mechanisms of asthma and COPD. *Clinical Science*. 2017; 131

7. Fitzpatrick, A. M. Biomarkers of asthma and allergic airway diseases. *Annals of Allergy, Asthma and Immunology*. 2015; 115(5), 335–40.

8. Nadeem, A., Siddiqui, N., Alharbi, N. O., & Alharbi, M. M. Airway and systemic oxidant-antioxidant dysregulation in asthma: A possible scenario of oxidants spill over from lung into blood. *Pulmonary Pharmacology and Therapeutics*. 2014; 29(1).

9. Nadeem, A., Chhabra, S. K., Masood, A., & Raj, H. G. Increased oxidative stress and altered levels of antioxidants in asthma. *Journal of Allergy and Clinical Immunology.* 2003; 111(1).

10. Barnes, P. J., & Drazen, J. M. Pathophysiology of Asthma. *Asthma and COPD*. 2002; 343–59.

11. Kumar, R. K., Herbert, C., & Foster, P. S. The "classical" ovalbumin challenge model of asthma in mice. *Current Drug Targets.* 2008; 9(6).

12. Kumar, R. K., Herbert, C., Webb, D. C., Li, L., & Foster, P. S. Effects of anticytokine therapy in a mouse model of chronic asthma. American *Journal of Respiratory and Critical Care Medicine.* 2004; 170(10), 1043–8.

13. Nials, A. T., & Uddin, S. Mouse models of allergic asthma: Acute and chronic allergen challenge, *DMM Disease Models and Mechanisms*. 2008; 1

14. Lee, M. Y., Seo, C. S., Lee, N. H., Ha, H., Lee, J. A., Lee, H., Lee, H., & Lee. K. Anti-asthmatic effect of schizandrin on OVA-induced airway inflammation in a murine asthma model. *International Immunopharmacology*. 2010; 10(11).

15. Zosky, G. R., & Sly, P. D. Animal models of asthma. *Clinical & Experimental Allergy.* 2007; 37(7), 973–88.

16. Chapman DG, Irvin CG, Kaminsky DA, Forgione PM, Bates JHT, Dixon AE. Influence of distinct asthma phenotypes on lung function following weight loss in the obese. *Respirology*. 2014; 19(8), 1170–7.

17. Sagar, S., Akbarshahi, H., & Uller, L. Translational value of animal models of asthma: Challenges and promises, *European Journal of Pharmacology*. 2015; 759

18. Miyake, T., Miyake, T., Sakaguchi, M., Nankai, H., Nakazawa, T., & Morishita, R. Prevention of Asthma Exacerbation in a Mouse Model by Simultaneous Inhibition of NF-κB and STAT6 Activation Using a Chimeric Decoy Strategy. *Molecular Therapy Nucleic Acids.* 2018; 10.

19. Aloamaka, O. E. O., Eiya, B. O., Aikpitanyi-Iduitua, R. O., Ighodaro, C. N., Ijewere, P. O., Ifeanyi, N. J., Esinulo, K. C., Obodo, F. O., Nweje, O. K., Omasaye, E. O., Tobalu, F. O., & Ezeuko, V. C. Optimising ovalbumin (OVA) sensitisation protocols for asthma modelling: frequency and duration effects on lung biomarkers. *Dutse Journal of Pure and Applied Sciences.* 2025; 11(3c SE-Articles), 360–71.

20. Bai, F., Fang, L., Hu. H., Yang, Y., Feng, X., & Sun, D. Vanillic acid mitigates the ovalbumin (OVA)-induced asthma in rat model through prevention of airway inflammation. Bioscience, Biotechnology, and Biochemistry. 2019; 83(3), 531–7.

21. Wu, W., Li, Y., Jiao, Z., Zhang, L., Wang, X., & Qin, R. Phyllanthin and hypophyllanthin from Phyllanthus amarus ameliorates immune-inflammatory response in ovalbumin-induced asthma: role of IgE, Nrf2, iNOs, TNF-α, and IL's. *Immunopharmacology and Immunotoxicology.* 2019; 41(1), 55–67.

22. Aloamaka, O. E. O, & Ighodaro, C. N. Assessing the Hepatic Effect of Common Antiasthmatic Medications (Montelukast, Prednisolone, Hydrocortisone, and Salbutamol) in a Sprague Dawley Rat Model of Asthma. *Journal of Experimental and Clinical Anatomy*. 2025; 22(3), 576–81.

23. Buege, J. A., & Aust, S. D. Microsomal Lipid Peroxidation. *Methods Enzymology.* 1978; 52(C).

24. Cohen, G., Dembiec, D., & Marcus, J. Measurement of catalase activity in tissue extracts. *Analytical Biochemistry. 1*970; 34(1).

25. Misra, H. P., & Fridovich, I. The role of superoxide anion in the autoxidation of epinephrine and a simple assay for superoxide dismutase. *Journal of Biological Chemistry.* 1972; 247(10).

26. Nyman, M. Serum haptoglobin. *Methodological and clinical studies*. 1959; 169-pp.

27. Bradford, M. M. A rapid and sensitive method for the quantitation of microgram quantities of protein utilizing the principle of protein-dye binding. *Analytical Biochemistry*. 1976; 72(1–2).

28. Fitzpatrick, A. M., Teague, W. G., Meyers, D.A., Peters, S. P., Li, X., Li, H., Wenzel, S. E., Aujla, S., Castro, M., Bacharier, L. B., Gaston, B. M., Bleecker, E. R., & Moore, W. C. National Institutes of Health/National Heart, Lung, and Blood Institute Severe Asthma Research Program. Heterogeneity of severe asthma in childhood: Confirmation by cluster analysis of children in the National Institutes of Health/National Heart, Lung, and Blood Institute Severe Asthma Research Program. *Journal of Allergy and Clinical Immunology*. 2011; 127(2).

29. Hamid, Q., Tulic', M. K., Liu, M. C., & Moqbel, R. Inflammatory cells in asthma: Mechanisms and

implications for therapy. In: *Journal of Allergy and Clinical Immunology*. 2003.

30. Shah, A. R., Chaudhari, S. N., & Shah, M. H. Role of Platelet Parameters in Diagnosing Various Clinical Conditions. *National Journal of Medical Research*. 2013; 3.
31. Ayala, A., Muñoz, M. F., & Argüelles, S. Lipid peroxidation: Production, metabolism, and signaling mechanisms of malondialdehyde and 4-hydroxy-2-nonenal, *Oxidative Medicine and Cellular Longevity*. 2014; 2014
32. Valko, M., Leibfritz, D., Moncol, J., Cronin, M. T. D., Mazur, M., & Telser, J. Free radicals and antioxidants in normal physiological functions and human disease, *International Journal of Biochemistry and Cell Biology*. 2007; 39
33. Zelko, I. N., Mariani, T. J., & Folz, R. J. Superoxide dismutase multigene family: A comparison of the CuZn-SOD (SOD1), Mn-SOD (SOD2), and EC-SOD (SOD3) gene structures, evolution, and expression, *Free Radical Biology and Medicine*. 2002; 33
34. Lubos, E., Loscalzo, J., & Handy, D. E. Glutathione peroxidase-1 in health and disease: From molecular mechanisms to therapeutic opportunities, *Antioxidants and Redox Signaling*. 2011; 15
35. Moore, K. W., De Waal Malefyt, R., Coffman, R. L., & O'Garra, A. Interleukin-10 and the interleukin-10 receptor, *Annual Review of Immunology*. 2001; 19
36. Hayden, M. S., & Ghosh, S. NF-κB in immunobiology, *Cell Research*. 2011; 21.

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