JOURNAL OF BIOMEDICINE AND BIOCHEMISTRY
Volume 4 Issue 4 (2025), pp. 66–75

ORIGINAL A R T I C A L

Journal JBB
Print ISSN:3006-0532
Online ISSN:2958-0528

Molecular and Clinical Perspectives on Cytokine Storms and Seasonal Influenza Infections

Wurood Kadhim Abed [1,*], Thualfakar Hayder Hasan Abed [2], Maha Mashrq AlBayati [3]

1,2 Jabir Ibn Hayyan University for Medical and Pharmaceutical Sciences, Al-Najaf 54001, Iraq
3 Department of Pathological Analyses, Faculty of Science, University of Kufa, Al-Najaf 54001,, Iraq.

Corresponding Author Email:
worood.k.abd@Jmu.edu.iq

Received: 20 September 2025,
Revised: 8 October 2025,
Accepted: 22 November 2025,
DOI: 10.57238/jbb.2025.7432.1156

OPEN ACCESS

Access this article online

ABSTRACT

Background: Seasonal influenza may cause severe complications in case of cytokine storm, a hyperinflammatory reaction that increases tissue damage and dysfunction of multiple organs

Objective: The research aims were to determine the correlation between seasonal influenza and cytokine storm through clinical and molecular investigations.

Methods: A cross-sectional observational study was carried out on 120 patients with RT-PCR-confirmed seasonal influenza in December 2023-March 2024. ELISA was used to determine serum levels of IL-6, TNF-α, IFN- g, IL-1, and IL-18. Demographics and clinical information, such as ARDS, coagulopathy, acute kidney injury, CNS manifestations, etc., were documented. Statistical significance of p = 0.05 was used to test statistical significance with SPSS v26.0 (t-test, Mann-Whitney, Pearson/Spearman correlation).

Results: The patients with cytokine storm (n = XX) had much more serum cytokines than their uncomplicated influenza counterparts (IL-6: 142 ± 35 vs. 18 ± 6 pg/mL, p < 0.001; TNF-α a: 96 ± 22 vs. 15 ± 5 pg/mL, p < 0.001). The incidence of coagulopathy (70% vs. 5%), AKI (40% vs. 5%), and CNS (confusion 45% vs. 10%) was significantly higher. High IL-6 levels were greatly associated with ARDS (r = 0.68, p < 0.01).

Conclusion: In seasonal influenza, cytokine storm is related to hyper-release of IL-6, TNF-α, IFN- gamma, IL- 1 8, and IL-1α, which cause dysfunction of multiple organs and increased complications. The timely identification of the signs of cytokine storm would enhance the outcome of patients and inform the use of specific immunomodulatory treatment

Keywords: Seasonal Influenza, cytokine storm, RIG-I / MAVS, RT-PCR, IL-6

1. Introduction

Against the background of seasonal influenza viruses, vaccines and antiviral therapy remain insufficient to mitigate the danger to global health[1]. Whereas the majority of the infections cause self-limiting illnesses, some cause severe ones that may cause hospitalization and death, especially in special groups: older individuals, young children, and people with other health complicationsp[6]. The severe influenza pathogenesis is not only due to viral multiplication but also largely due to the response of the host immune system, in particular, cytokine release syndrome (or cytokine storm) [5]. Cytokine storm. First used in 1993, the term cytokine storm was coined to refer to the engraftment syndrome that accompanied acute graft-versus-host disease that developed after allogeneic hematopoietic stem cell

transplantation [3]. Nevertheless, the idea of having an elevated immune reaction after developing systemic viral diseases had already been acknowledged previously, and it was called an "influenza-like syndrome" in 1958. Since that time, our awareness of cytokine storm has changed dramatically, especially in the arena of respiratory viral infections such as seasonal influenza[4][8][24]. Cytokine storm is diagnosed due to the overactive development of immune cells and a considerable rise in cytokine levels in circulation [14][34]. This disease condition has been associated with the pathogenesis of life-threatening diseases like acute respiratory distress syndrome (ARDS) that commonly occur as complications of severe influenza[22].

The immune dysregulation is characterized by complex mechanisms of interactions of numerous immune cells, cytokines, and chemokines, which result in a positive feedback loop that leads to worsening tissue destruction and organ failure [2][8]. The elucidation of molecular mechanisms of the interaction between seasonal influenza and cytokine storm is important in order to develop effective approaches to therapy [4].The purpose of this study is to show the relationship between seasonal influenza and cytokine storm.

2. Materials and Methods:

2.1. Study Design and Ethical Considerations

It is a cross-sectional observational study aimed at examining the correlation between the infection of the seasonal influenza virus and the development of the cytokine storm among affected subjects. The study received ethical clearance from the concerned review board, and informed consent was sought from the participants or their parents in writing.

2.2. Data Collection

The information was all gathered prospectively in the course of the influenza season between December 2023 and March 2024. A total of 120 blood samples were obtained from patients who had symptoms associated with influenza, as was evident during the season. In the public health hospitals and outpatient clinics, patients with clinical manifestations of the disease due to influenza infection (e.g., high fever, cough, myalgia, and sore throat) are seen. Once the RT-PCR (According to what was taken from hospitals and healthcare centres in Sari city in Mazandaran province in Iran) confirmed the influenza infection, the participants were recruited into the study.

Two types of samples were taken for the study: nasopharyngeal Swabs, to confirm by the molecular method (RT-PCR) the presence of the influenza virus Blood Samples: Venous samples to be taken to analyze cytokines and profile the immune system Volume Collected: 5 mL of whole blood from each patient The tubes used were EDTA tubes to separate plasma and plain tubes to separate serum [2,3].

2.3. Study Design:

This was a cross-sectional observational study conducted between December 2023 and March 2024. All data (demographics, laboratory results, and complications) were collected at the time of admission without follow-up. Progression to complications such as ARDS, AKI, or CNS manifestations was assessed based on the patient's hospital records during The study design is shown clearly in Figure 1.

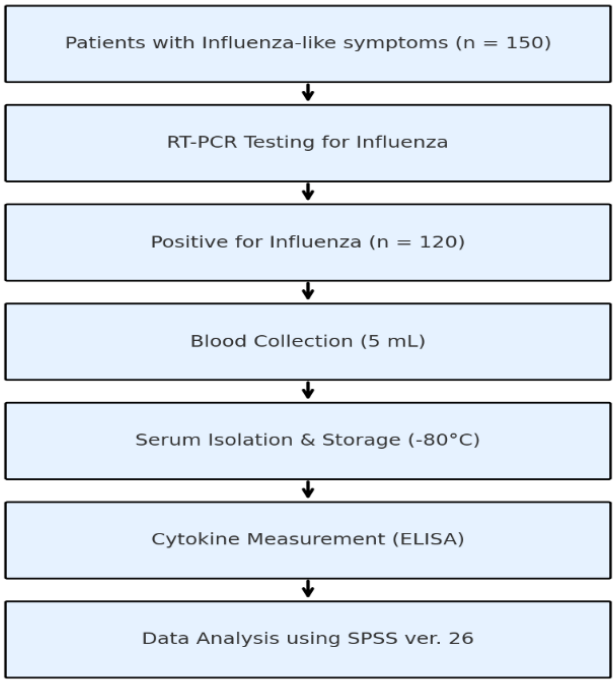

Figure 1. The workflow of the study presented in a schematic describes the stages of work

2.4. Inclusion Criteria and Exclusion Criteria:

- Inclusion Criteria

Patients who are >=18 years, Findings that are typical of influenza (e.g., fever, cough, sore throat, fatigue), Positive through RT-PCR seasonal influenza

- Exclusion Criteria:

People with autoimmune disorders or chronic inflammatory disorders, Patients using immunosuppressing medication[7].

2.5. Laboratory Methods

2.5.1. Identification of Influenza Viruses

Technique: Reverse Transcriptase Reaction Polymerase Chain Reaction (RT-PCR), RNA Extraction: Done by a commercial viral RNA extraction kit (human RNA extraction kit, Qiagen).

RT-PCR Kit: Into Influenza A and B, fluorescein-specific to the M and HA genes[8]

2.5.2. Thermal Cycler of Parameters:

In the following Table1, the thermal cycler conditions for the influenza virus will be shown

Table 1. Thermal cycles of RT.PCR for the influenza virus.

Steps	Temperature (˚C)	Time	Number of cycles
Reveres transcription	50 ˚C	30 minutes	1 cycle
Initial denaturation	95˚ C	10 minutes	1 cycle

Denaturation	95 ˚ C	15 seconds	40 cycles
Annealing \extension	60˚ C	1 minutes	40 cycles

2.5.3. Cytokine Quantification

Technique Employed: ELISA Enzyme-Linked Immunosorbent Assay

Cytokines Tested (Interleukin-6 (IL-6), Tumour Necrosis Factor-alpha (TNF-α), Interferon-gamma (IFN-γ), Interleukin-10 (IL-10). Commercial Kits: ELISA kits of respected suppliers (from R&D Systems as per the manufacturer's instructions. Sample Processing: The blood samples were centrifuged at 3000 rpm for 10 minutes, and the serum was retrieved and aliquoted, and maintained at -80 °C until the analysis date[10][11].

2.6. Data Analysis

Operating software: SPSS, v. 26.0 (IBM, USA)

Tests Used (Mean, median, SD, Mann-Whitney U-test or t-test of student approaches to comparisons, Pearson or Spearman correlation to determine associations between the level of cytokines and the severity of the influenza) Significance Level: Stage of statistical significance was used as p-value < 0.05[12].

3. Results and Discussion

3.1. Demographics

Table 2. The baseline characteristics of the study population (n = 120)

Variable	Total (n=120)	Uncomplicated influenza (n=80)	Cytokine storm (n=40)
Age (years, mean ± SD)	46.2 ± 15.3	42.8 ± 13.1	53.7 ± 17.4
Gender (M/F)	68 / 52	42 / 38	26 / 14
Diabetes mellitus (%)	25 (20.8%)	12 (15%)	13 (32.5%)
Hypertension (%)	29 (24.1%)	14 (17.5%)	15 (37.5%)
COPD/asthma (%)	18 (15%)	9 (11.2%)	9 (22.5%)
Severity at presentation	Mild: 60 (50%) Moderate: 40 (33%) Severe: 20 (17%)	Mostly mild/moderate	Mostly severe

3.2. Definition and Characteristics of Cytokine Storm in Seasonal Influenza

The chart shows clearly that every mentioned clinical manifestation is more common in patients with a cytokine storm than in patients with seasonal influenza of an uncomplicated course. The most striking increase was observed in coagulopathy (14×), with an increase in frequency of 5 and 70% in seasonal influenza and cytokine storm association, respectively. Other indicators of multi-organ dysfunction, such as lymphopenia, hypotension, and acute kidney injury, were also significantly increased. Confusion/Delirium increased 4.5 fold; thus, CNS may be involved as a result of the overreaction of cytokine release, as shown Figure 2.

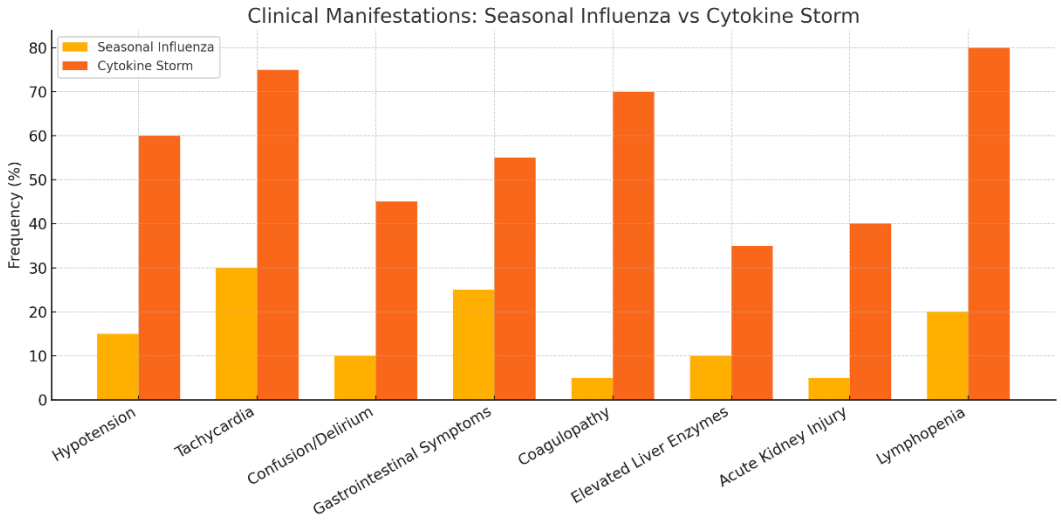

Figure 2. Characteristics of Cytokine Storm in Seasonal Influenza

The information contributes to the consideration of cytokine storm as a contributor to systemic complications of severe cases of influenza. Most cases of seasonal influenza run a self-limiting course; however, there are subsets of these cases that develop into a hyperinflammatory state (e.g., excess cytokine production of IL-6, TNF-α, IFN-γ). This leads to Leakage and hypotension (in 60% vs 15%), Heart stress and tachycardia, Dysfunction of the CNS (Confusion/Delirium: 45 % vs 10), Lymphopenia indicates exhaustion of the immune response, and is one of the characteristics of heavily infected viruses[13].

Found in a study found that cytokine storms cause immune dysregulation and multi-organ failure, which increases the mortality rates during influenza pandemics, including the H1N1 pandemic [27]. Recently highlighted that under the influence of influenza A, high pro-inflammatory cytokines are associated with ARDS, coagulopathies, and hepatic injury in the case of influenza A infection[33]. Such a profound rise is indicative of systemic vasodilation and capillary leak syndrome caused by the release of excessive cytokines, mainly TNF-α and IL-6, resulting in hemodynamic instability and the risk of shock[10][16][22][32].

Tachycardia occurring during cytokine storms is commonly caused by fever, hypoxia, and hypotensive compensation. The inflammatory condition on a systemic level increases the cardiac workload and strain[7][9]. Such cytokines as IL-6 and TNF-alpha cause damage to mucosal tissue and cause nausea, vomiting, and diarrhoea. Moreover, the symptoms can represent a systemic inflammation involving the GI tract[11][13][22].

It is one of the most important discoveries. Cytokine storm is also activated in coagulation pathways (specifically through the expression of IL-6 and tissue factor), creating states similar to DIC or thrombotic complications[16]). Injury to the liver follows hypoperfusion, as well as an inflammatory hepatocellular injury of direct nature, driven by an upsurge of circulating cytokines (IL-1, IL-6) [24]. The most common complication is AKI, which is caused by renal hypoperfusion with the help of cytokines, inflammation of the renal tissues, and microthrombi. This is why it increased 8 times[18]. Severe viral illness is characterized by lymphopenia. It is due to apoptosis caused by the excessive proportions of IFN- A and IL-6. It denotes one of the markers of immune exhaustion, which can be followed by poor outcomes[19].

3.3. Molecular Mechanisms of Cytokine Storm in Seasonal Influenza

The pathogenesis of cytokine storm in seasonal influenza begins with the recognition of viral components by the host's immune system. This study found a difference between seasonal influenza and influenza in which a cytokine storm occurs, as shown in Figure 3.

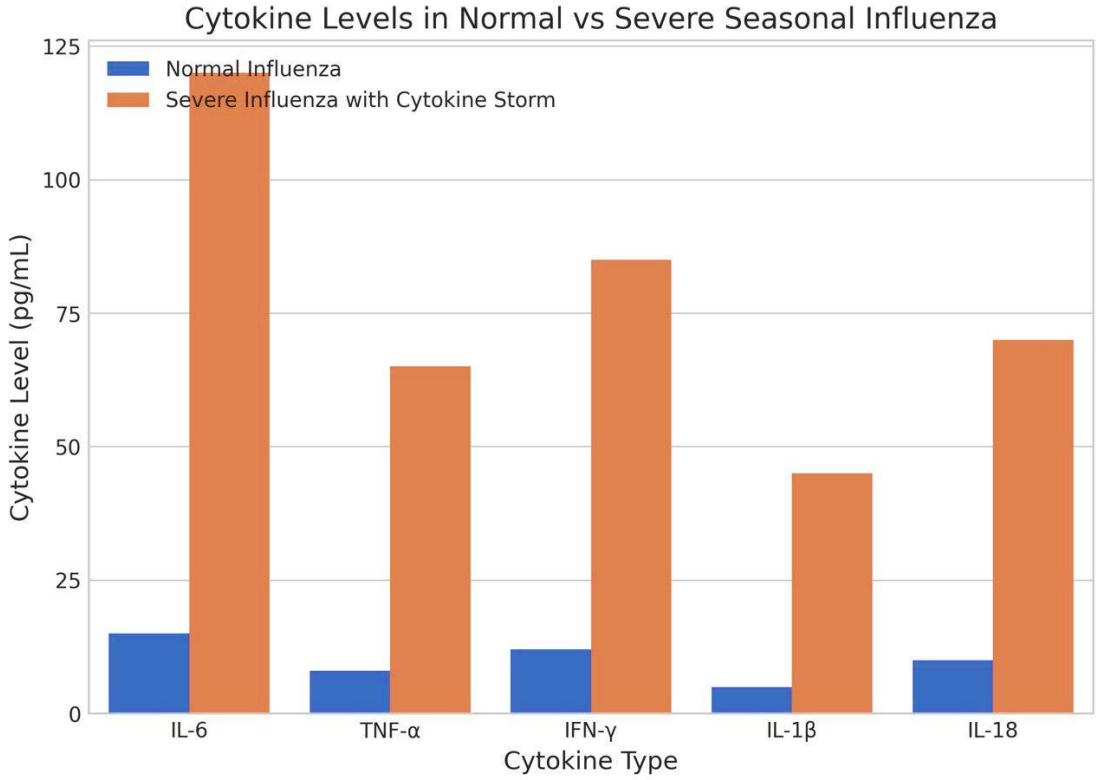

Figure 3. The serum levels of cytokines in patients with influenza with no complications and cytokine storm*Error bars represent mean ± SD. Statistically significant (t-test, p = 2. 0 x 10), 2 = -3. 1 2, r = 0.455. Cytokine storm patients had a significant increase in IL-6, TNF-alpha, IFN-gamma, IL-1 beta, and IL-18.

IL-6 is a key factor in cytokine Storms. It is also involved in fever, vascular permeability, and the synthesis of acute-phase proteins (Table 3). The almost 8-fold increase in severe cases correlates with hyperactivation of the immune system and pulmonary inflammation, with a bad outcome (Levi & van der Poll, 2022).

Table 3. Serum cytokine levels (mean ± SD, pg/mL)

Cytokine	Uncomplicated influenza (n=80)	Cytokine storm (n=40)	p-value
IL-6	18 ± 6	142 ± 35	<0.001
TNF-α	15 ± 5	96 ± 22	<0.001
IFN-γ	12 ± 4	78 ± 18	<0.001
IL-1β	10 ± 3	62 ± 15	<0.001
IL-18	14 ± 5	85 ± 21	<0.001

One of the first and most frequently raised cytokines on influenza-mediated hyperinflammatory reactions is IL-6. Its production involves the alveolar macrophages, epithelial and endothelial cells, in response to RNA produced by the virus through RIG-I / MAVS and TLR. Normally, IL-6 facilitates the coordination of immunity and the production of acute-phase proteins ([24][28]. TNF-α leads to endothelial activation, vasodilation, and contributes to hypotension and multi-organ failure. An increased level of TNF-alpha is an indication of systemic inflammation as well as coagulopathy, especially among the critically ill patients [1]. TNF-alpha is largely in charge of the orchestration of systemic inflammation. Monocytes and macrophages secrete it very quickly after detecting the viral RNA. Whereas low levels augment the traffic of immune cells and the elimination of viruses at an early stage, high levels of TNF-alpha foster[15].

Primarily, the NK and T-cells produce IFN-γ, and this helps in the major antiviral protection. Nevertheless, when elevated, there is tissue damage and immune exhaustion, particularly in the lung parenchyma. The respective extreme increase indicates excessive immunity of the cell in cytokine storms (Zheng et al., 2025). NK, Th1 cells, and CD8+ + T-cells secrete IFN-gamma. It plays a crucial role in the clearance of viruses, upregulation of MHC-I, and activation of macrophages. But during a cytokine storm, high concentrations of IFN-γ are maintained mainly [3][31].

Following inflammasome activation, IL-1beta is released and causes fever, leukocyte migration, and tissue destruction. Since its increase in cytokine storm indicates active participation in the NLRP3 inflammasome, it has the characteristic of severe influenza[17][30].

The production of IL-1beta is through the activation of the inflammasome (mainly NLRP3) and exerts a potent pyrogenic effect as well as a pro-inflammatory capacity. In a cytokine storm, the IL-1beta increases the recruitment of neutrophils, causing damage to the alveolar-capillary barriers [31]. It enhances endothelial activation, which continues vascular leakage. Has a synergistic effect with IL-6 that aids in systemic inflammation. Antagonists of the IL-1 signalling have led to protective effects in the murine model of lung injury due to influenza infection[26]. Another cytokine that is related to the inflammasome is IL-18.

It triggers the IFN-γ production and promotes the activity of a cytotoxic lymphocyte [29]. In a cytokine storm, as in influenza The overproduction of IL-18 enhances inflammation and apoptosis of the epithelium in the lungs high viral burden is associated with IL-18, and the mortality was related to it enhances the activity of IL-12 and IL-15 known to stimulate hypercytotoxic T-cells responses New evidence indicates that IL-18 blockade could be a forms of a new targeted immunomodulatory treatment in cytokine storm syndromes[20][21]. IL-18 is inflammasome-dependent as IL-18. It intensifies the production of IFN- and increases the speed of the immune cell cytotoxicity. This will promote epithelial injuries and systemic inflammation in the lungs. A high level of IL-18 is a bad prognosis factor [23][25].

4. Conclusion:

Seasonal influenza may become a mild respiratory infection that develops an acute systemic syndrome that threatens to take lives, upon complicated by cytokine storm. The results of this research present the molecular pathogenesis, clinical manifestations, and remedial prospects of this severe immunopathological condition. Identifying and treating cytokine storm in an early stage and using a multiple approach to therapy can be the means of much less morbidity and mortality.

Acknowledgements: We express our profound gratitude to Jabir Ibn Hayyan University for Medical and Pharmaceutical Sciences, Najaf, Iraq, for their support

Ethical Statement: The study protocol of the sample collection of human material was determined by the Ethical Committee of Jabir Ibn Hayyan University of Medical and Pharmaceutical Sciences, Najaf,

Iraq. Every process was done in accordance with the stipulated guidelines and all the actions and considerations required to safeguard the welfare and the rights of the human subject were put in place.

Competing interest declaration: All the authors of this manuscript report that there are no financial or personal relations to any other individual or agency that can improperly interfere with the intellectual work contained in our paper.

Acknowledgements: We express our profound gratitude to Jabir Ibn Hayyan University for Medical and Pharmaceutical Sciences, Najaf, Iraq, for their support

Ethical Statement: The study protocol of the sample collection of human material was determined by the Ethical Committee of Jabir Ibn Hayyan University of Medical and Pharmaceutical Sciences, Najaf, Iraq. Every process was done in accordance with the stipulated guidelines and all the actions and considerations required to safeguard the welfare and the rights of the human subject were put in place.

Competing interest declaration: All the authors of this manuscript report that there are no financial or personal relations to any other individual or agency that can improperly interfere with the intellectual work contained in our paper.

Conflict of interest: The authors have no conflict of interest.

The manuscript has been agreed upon by all authors to be submitted.

Funding: This study did not obtain any funding.

References:

1. Abed WK, Hussein HK. Detection of human metapneumovirus RNA sequence in nasopharyngeal swab samples from children with acute respiratory tract infections in Najaf Province, Iraq. AIP Conf Proc. 2023;2977:040009. https://doi.org/10.1063/5.0182237
2. Abusaiba THH. Prevalence of hepatitis B and C viruses in patients in Al-Najaf Governorate, Iraq. Int J Pharm Res. 2020;12(3):1297–1303.https://doi.org/10.31838/ijpr/2020.12.03.195
3. Al-Ethari AS, Hasan TH, Tikki KA, Bustani GS. Genotypic detection of qnrA and qnrC genes in Citrobacter koseri isolated from patients with urinary tract infection. Razi Arch Inst. 2022;77(2).https//doi.org/10.22092/ARI.2022.357122.1979.
4. Aljanaby IAJ, Al-Labban HMY, Hayder T, Hasan MJA, Aljanaby AAJ. Neural cell adhesion molecule in Mycobacterium tuberculosis patients. Lat Am J Pharm. 2023;42(Spec Issue):184–187.
5. Blanco-Melo D, Nilsson-Payant BE, Liu WC, Uhl S, Hoagland D, Møller R, et al. Imbalanced host response to SARS-CoV-2 drives development of COVID-19. Cell. 2020;181(5):1036–1045.e9. https://doi.org/10.1016/j.cell.2020.04.026
6. Chakraborty C, Bhattacharya M, Das A, Saha A. Regulation of miRNA in cytokine storm of COVID-19 and other viral infections: an exhaustive review. Rev Med Virol. 2025;35(2):e70026. https://doi.org/10.1002/rmv.70026
7. El-Maradny Y, Othman A, Gerges M, Belal F, Behery E, El-Fakharany E. COVID-19 coronavirus: pathogenesis, clinical features, diagnostics, epidemiology, prevention and control. Microb Biosyst. 2020;5(1):7–20. https://doi.org/10.21608/mb.2020.33405.1018
8. Freeman TL, Swartz TH. Targeting the NLRP3 inflammasome in severe influenza. Front Immunol. 2020;11:1159. https://doi.org/10.3389/fimmu.2020.01159

9. Goenka A, Michael BD, Ledger E, Hart IJ, Absoud M, Chow G, et al. Neurologic complications of influenza infection. J Pediatric Infect Dis Soc. 2021;10(1):46–53. https://doi.org/10.1093/jpids/piaa098
10. Gu Y, Zuo X, Zhang S, Ouyang Z, Jiang S, Wang F, et al. The mechanism behind influenza virus cytokine storm. Viruses. 2021;13(7):1362. https://doi.org/10.3390/v13071362
11. Guo M, Ye YD, Cai JP, Xu HT, Wei W, Sun JY, et al. PEG-SeNPs as therapeutic agents inhibiting apoptosis and inflammation of cells infected with H1N1 influenza A virus. Sci Rep. 2024;14(1):21318. https://doi.org/10.1038/s41598-024-71898-0
12. Hasan TH. Extended spectrum beta-lactamase Escherichia coli isolated from UTI patients in Najaf Province, Iraq. Int J Pharm Res. 2020;12(4):673–677.https://doi.org/10.31838/ijpr/2020.12.04.049
13. Hasan TH, Al-Harmoosh RA, Al-Khilkhali HJB. Identification of HIV virus in Najaf City, Iraq. Int J Res Pharm Sci. 2020;11(3):4866–4871.
14. Hasan TH, Kadhum HA, Al-Khilkhali HJB. Epidemiology of varicella-zoster virus in Najaf Governorate, Iraq. Lat Am J Pharm. 2023;42(Spec Issue):188–190.
15. Hassan LA, Darweesh MF, Hasan TH. Immunomodulator activity of Pseudomonas aeruginosa extracted protein: azurin charming protein. Lat Am J Pharm. 2023;42(Spec Issue):245–251.
16. Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020;395(10223):497–506. https://doi.org/10.1016/S0140-6736(20)30183-5
17. Hussein AR, Al-Challabi K, Abed W, Mahdi H, Al-Hatimy A. Correlation of cytokines with thyroid hormones and autoantibodies in thyroid autoimmune diseases. Egypt J Med Microbiol. 2025;34(1):99–109. https://doi.org/10.21608/ejmm.2024.322552.1344
18. Iwasaki A, Pillai PS. Inflammatory responses and liver injury in viral infection. J Hepatol. 2021;75(3):712–722. https://doi.org/10.1016/j.jhep.2021.04.031
19. Kazantseva ED, Petrova AG, Rychkova LV, Darenskaya MA. Cytokines in influenza in children: a literature review. Cell Tissue Biol. 2024;18(6):632–645.
20. Lee YJ, Park SY, Kim YK, Kim S. Cytokine profiles in severe influenza A. Front Immunol. 2023;14:1134572. https://doi.org/10.3389/fimmu.2023.1134572
21. Levi M, van der Poll T. Coagulation and sepsis. N Engl J Med. 2022;387(1):77–88. https://doi.org/10.1056/NEJMra2107772
22. Lv C, Guo J, Luo R, Li Y, Zou X, Wang T, et al. Taurolidine inhibits influenza virus infection and prevents influenza-induced cytokine storm, vasoconstriction and lung damage. Cell Mol Life Sci. 2025;82(1):1–18.https://doi.org/10.1007/s00018-025-05636-6
23. Majeed HT, Hasan TH, Aljanaby AAJ. Epidemiological study in women infected with Toxoplasma gondii, rubella virus, and cytomegalovirus in Al-Najaf Governorate, Iraq. Int J Pharm Res. 2020;12:1442–1447.
24. Morris G, Bortolasci CC, Puri BK, Marx W, O'Neil A, Athan E, et al. Cytokine storms of COVID-19, H1N1 influenza, CRS and MAS compared. Cytokine. 2021;144:155593. https://doi.org/10.1016/j.cyto.2021.155593
25. Murdaca G, Paladin F, Tonacci A, Isola S, Allegra A, Gangemi S. Potential role of cytokine storm pathway in viral respiratory pandemics. Biomedicines. 2021;9(11):1688.

https://doi.org/10.3390/biomedicines9111688

26. Olbei M, Hautefort I, Modos D, Treveil A, Poletti M, Gul L, et al. SARS-CoV-2 causes a different cytokine response compared to other respiratory viruses. Front Immunol. 2021;12:629193. https://doi.org/10.3389/fimmu.2021.629193
27. Shlash SA, Kadhum HA, Hasnawi NM, Hasan TH. Environmental and parasitic contamination of Euphrates River water using granular activated date seeds. Mater Today Proc. 2023;80:3646–3654.
28. Shlash SA, Zwain KA, Khayoon SQ, Hasan TH. Vitamin D deficiency with Blastocystis hominis infection. AIP Conf Proc. 2023;2845(1). https://doi.org/10.1063/5.0157297
29. Stegeman SK, Kourko O, Amsden H, Pellizzari Delano IE, Mamatis JE, Roth M, et al. RNA viruses, TLRs, and cytokines: the perfect storm? J Innate Immun. 2025;39:1–?
30. Teijaro JR. Cytokine storms in infectious diseases. Semin Immunopathol. 2016;38(5):513–515. https://doi.org/10.1007/s00281-016-0559-1
31. Tikki KA, Mohammed EH, Mahdi AA, Hasan TH. Effects of COVID-19 infection on hematologic parameters. Egypt J Hosp Med. 2022;89(1):5488–5492. https://doi.org/10.21608/ejhm.2022.264846
32. Wang S, Wang D, Wang X, Chen M, Wang Y, Zhou H, et al. Cytokine storm related to CD4+ T cells in influenza-associated acute necrotizing encephalopathy. Immune Netw. 2024;24(2):e18.https://doi.org/ 10.4110/in.2024.24.e18
33. Yin YZ, Luo RL, Li SQ, Li RF. Mechanism and suggestions of liver damage caused by influenza A virus: a narrative review. Infect Dis Trop Med. 2024;10:e1392.https://doi.org/ 10.32113/idtm_20244_1392
34. Yuan S, Jiang SC, Zhang ZW, Fu YF, Hu J, Li ZL. Quantification of cytokine storms during virus infections. Front Immunol. 2021;12:659419. https://doi.org/10.3389/fimmu.2021.659419
35. Zheng Y, He D, Zuo W, Wang W, Wu K, Wu H, et al. Influenza A virus dissemination leads to tissue-resident cell injury and dysfunction in viral sepsis. EBioMedicine. 2025;116:104715.https://doi.org/ 10.1016/j.ebiom.2025.105738

How to cite this article

Abed WK, Abed THH, AlBayati MM. Molecular and Clinical Perspectives on Cytokine Storms and Seasonal Influenza Infections. Journal of Biomedicine and Biochemistry. 2025;4(4):66-75. doi:10.57238/jbb.2025.7432.1156