WHIOCE PUBLISHING PTE. LTD.
PROVIDING FIRST-CLASS SCIENTIFIC INFORMATION FOR TOP SCHOLARS

2025 Volume 10, Issue 3
Advances in Precision Medicine
ISSN: 2424-9106 (Online)
ISSN: 2424-8592 (Print)

Diabetes with Viral Pneumonia: Epidemiological Characteristics, Pathophysiological Mechanisms, and Clinical Management Strategies

Zhengmei Qian, Xiaoxuan Chen, Ruilin Sun*
The Affiliated Guangdong Second Provincial General Hospital of Jinan University, Guangzhou 510317, Guangdong, China

**Author to whom correspondence should be addressed.*

***A*bstract:** Diabetes is one of the susceptibility risk factors for viral pneumonia, particularly during COVID-19 pandemic. Patients with diabetes are more likely to develop severe disease following infection and face a markedly increased risk of complications, substantially increasing the healthcare burden. The primary mechanism involves persistent hyperglycemia impairing immune system function through multiple pathways, promoting excessive inflammatory responses, and thereby exacerbating lung tissue damage. Research indicates that effective glycemic control and vaccination can substantially reduce mortality rates and the risk of severe disease progression in diabetic patients with viral pneumonia. This review aims to summarize the clinical associations, immunopathological mechanisms, and prevention strategies related to diabetes and viral pneumonia, providing a theoretical basis for improving patient outcomes.
Keywords: COVID-19; Diabetes; Influenza virus; Pneumonia

Online publication: September 26, 2025

1. Background

Data from the Global Burden of Disease study indicate that diabetes and lower respiratory infections are both major threats to human health. In 2019, diabetes ranked as the eighth leading cause of death globally, while lower respiratory infections ranked fourth. With the outbreak of the COVID-19 pandemic, coronavirus infections surged to become the second leading cause of death worldwide in 2021 [1].

Diabetes, a group of chronic metabolic disorders caused by the combined effects of genetic, environmental, and lifestyle factors, is characterized by persistent hyperglycemia resulting from impaired insulin secretion or action. Individuals with diabetes constitute a high-risk population for respiratory viral infections, exhibiting significant synergistic pathogenic effects. This interaction manifests not only as increased susceptibility to infection but also as markedly worsened clinical outcomes, creating a vicious cycle from infection to adverse events.

This review examines the disease burden, epidemiological characteristics, pathophysiological mechanisms, and clinical management strategies for viral pneumonia in diabetic patients. It aims to guide clinicians in early recognition,

precise diagnosis, and timely intervention, thereby reducing mortality in severe cases and alleviating the overall disease burden.

2. Epidemiological characteristics

2.1. Disease burden of diabetes in China

With changes in lifestyle and dietary habits, diabetes has become the third leading factor affecting human health after cardiovascular and cerebrovascular diseases and malignant tumors [2]. In 2021, approximately 530 million people worldwide had diabetes. By 2050, this number is projected to more than double, reaching around 1.3 billion people, with a prevalence rate approaching 10%—far exceeding the 6% rate recorded in 2021 [3]. In China, approximately 233 million people have diabetes, accounting for about one-quarter of the global diabetic population. More alarmingly, without effective interventions, China's diabetes prevalence is projected to climb to 29.1% by 2050, meaning nearly one-third of the Chinese population will have diabetes [4].

This massive patient population imposes a heavy economic burden. Research projects that from 2020 to 2030, diabetes-related healthcare costs in China will increase from $250.2 billion to $460.4 billion, while the proportion of total diabetes costs relative to GDP will rise from 1.58% to 1.69%. This indicates that the economic burden of diabetes will grow faster than China's economic growth rate [5].

2.2. Susceptibility of diabetes patients to viral pneumonia

Diabetic patients are prone to various infectious diseases due to prolonged hyperglycemia, metabolic disorders, and compromised immune function, with pulmonary infections being a common complication. Relevant studies indicate that pneumonia accounts for 26% of hospitalizations among diabetic patients and constitutes 8% of direct causes of death in advanced diabetes [6]. Previous research on diabetes in viral-induced pulmonary infections has been limited.

The 2020 novel coronavirus outbreak highlighted diabetes' significant role in viral infections. Epidemiological evidence indicates that type 2 diabetes is the second most common comorbidity among COVID-19 patients [7]. Clinical studies specifically demonstrate that poorly controlled diabetic patients face higher risks of adverse outcomes and mortality after SARS-CoV-2 infection compared to the general COVID-19 population. A Wuhan-based study found that among 1,561 COVID-19 patients enrolled, 153 had diabetes, representing a prevalence of 9.8% [8]. The study also revealed that diabetic patients exhibited higher mortality rates following SARS-CoV-2 infection.

3. Pathophysiological mechanisms

3.1. Increased viral susceptibility and impaired host defense barriers

SARS-CoV-2 infects host cells via the angiotensin-converting enzyme 2 (ACE2) receptor. Elevated levels of ACE2 receptors on cell membranes have been observed in diabetic patients, potentially explaining their heightened susceptibility to SARS-CoV-2 infection [9,10,11]. Hyperglycemia in diabetic patients elevates glycated hemoglobin (deoxygenated form) levels in red blood cells [10,11,12].

The surface protein of SARS-CoV-2 attacks the 1-beta chain of hemoglobin, and deoxyhemoglobin is more vulnerable than oxyhemoglobin, leading to the body not receiving enough oxygen, ultimately resulting in symptoms of respiratory distress. This impairs oxygen delivery to tissues, ultimately leading to respiratory distress. Hyperglycemia also induces structural changes in lung tissue, including partial lung collapse and increased vascular permeability [10,11] leading to pulmonary dysfunction. This impaired lung function in diabetic patients further accelerates SARS-CoV-2 viral invasion, exacerbating disease progression.

3.2. Immunological dysfunction

Viruses exploit the enhanced glycolytic pathway in host cells to obtain energy and raw materials for replication. A hyperglycemic environment forces key innate immune cells (such as monocytes, macrophages, and dendritic cells) to undergo metabolic reprogramming, rendering them overly reliant on glycolysis. This leads to multiple mechanisms—including lactate-mediated signaling inhibition, acetylation-related epigenetic disruption, and protein dysfunction caused by lipid peroxidation [13-15]. These synergistic effects impair innate immune cell functions in pathogen recognition, signal transduction, and antigen presentation, significantly weakening the host's antiviral defenses [13-17].

Metabolic dysregulation induced by hyperglycemia, particularly lipid peroxidation (LPO) resulting from abnormal lipid metabolism, directly suppresses T cell activation and function, leading to failure of the virus-specific immune response. The core mechanism involves a high-sugar environment triggering reactive oxygen species (ROS) accumulation and polyunsaturated fatty acid (PUFA) buildup within $CD4^+$ T cells, initiating a chain reaction of LPO. The active aldehyde compounds produced by LPO (e.g., 4-HNE) carbonylate STAT4, a key transcription factor for Th1 cell differentiation, leading to its ubiquitination and degradation. The loss of STAT4 impairs downstream T-bet expression and drastically reduces IFN-γ production, ultimately causing the complete collapse of the Th1 differentiation program [17].

Concurrently, high glucose induces acetyl-CoA accumulation and excessive histone acetylation, disrupting antigen presentation by dendritic cells (DCs) and indirectly weakening T cell activation [15]. These metabolic-immune crosstalk dysregulations collectively cause failure of virus-specific immune responses, delaying viral clearance and exacerbating disease severity in infected individuals.

3.3. Dysfunction of the immune-endocrine regulatory circuit

During the initial phase of severe viral infection, the body initiates a finely tuned "immune-endocrine" regulatory circuit to coordinate systemic metabolism and optimize antiviral defense [16]. Its core mechanism involves the rapid activation of γδ T cells residing in tissues such as the lungs and splenic red pulp, which become an important early source of interferon-γ (IFNγ). Systemically released IFNγ strongly stimulates insulin synthesis and secretion by activating IFNγ receptors on pancreatic β-cell surfaces and their downstream JAK-STAT signaling pathways. The abrupt elevation of insulin levels exerts potent hypoglycemic effects: it inhibits hepatic gluconeogenesis and glycogenolysis while promoting glucose uptake and utilization in peripheral tissues, leading to transient physiological hypoglycemia.

This self-induced "glucose restriction" environment provides dual critical protective effects for the host as follows:

(1) It directly limits the replication of many viruses highly dependent on glycolysis for energy, cutting off their energy and biosynthetic raw material supply;

(2) It alleviates lactic acid accumulation caused by excessive glycolysis, thereby releasing the inhibition of key innate signaling pathways like interferon regulatory factor 3 (IRF3) and nuclear factor κB (NF-κB) .

This significantly enhances the production of type I interferons (IFN-I), establishing a more robust systemic antiviral state. However, in diabetic patients—particularly those with type 1 diabetes or advanced type 2 diabetes where β-cell function is nearly depleted—this protective circuitry fails completely. The core defect lies in pancreatic β-cells' inability to respond to IFNγ signals and secrete sufficient insulin. Without insulin—the critical effector molecule—the liver's glucose output remains unchecked. Persistently elevated blood glucose levels prevent the induction of a beneficial "hypoglycemic window." This not only provides ample fuel for viral replication, leading to substantially increased viral load, but also perpetuates lactate-mediated immunosuppression. Ultimately, this results in impaired IFN-I responses, uncontrolled viral replication, and a heightened susceptibility to severe disease progression.

3.4. Tissue barrier disruption and inflammatory storm

Hyperglycemia directly impairs tissue barrier function through multiple pathways. Persistent hyperglycemia induces "glucotoxicity" in vascular endothelial cells and alveolar epithelial cells, triggering excessive production of mitochondrial reactive oxygen species (mtROS). This leads to cellular DNA damage and apoptosis, subsequently compromising the

integrity of intercellular junctions and basement membranes [17]. This compromised barrier function facilitates viral invasion and spread while exacerbating tissue edema and inflammatory exudation.

Concurrently, dysfunctional innate immune cells (e.g., alveolar macrophages) undergo severe metabolic-immune dysregulation under hyperglycemic stimulation. They exhibit "glycolytic dominance", a metabolic reprogramming that drives their differentiation toward a dysfunctional inflammatory phenotype [13]. Its core characteristics include high production of reactive oxygen species (ROS) and excessive pro-inflammatory cytokines (e.g., IL-6, IL-1β, TNF-α), yet an inability to effectively initiate critical antiviral programs.

This is primarily due to high glucose/glycolysis-derived lactate accumulation, which directly inhibits the activation of IRF3 and NF-κB signaling pathways. This leads to severe insufficiency in the production of core antiviral cytokines, including type I interferons (IFN-I) and type III interferons (IFN-λ). This "dysregulated inflammatory response" forms a vicious cycle [13]. On one hand, excessive ineffective proinflammatory factors recruit more immune cells to the lesion site, triggering a "cytokine storm." This excessive inflammation causes secondary damage to alveolar epithelium and vascular endothelium, dramatically increasing vascular permeability and leading to massive protein-rich fluid leakage—the classic pathological basis of acute respiratory distress syndrome (ARDS).

On the other hand, the absence of IFN-I fails to effectively suppress viral replication and dissemination within host cells, further exacerbating tissue damage. Ultimately, this immune disorder—incapable of clearing the virus while inflicting severe damage on the body's own tissues—can rapidly spread from the lungs to the entire body, leading to multiple organ dysfunction syndrome (MODS) [13].

4. Clinical characteristics and interactions: The bidirectional vicious cycle between diabetes and viral pneumonia

4.1. Diabetes exacerbates the severity of viral pneumonia

Across multiple viral pandemics, diabetes has consistently been identified as a key factor significantly increasing the risk of severe illness and death among patients [18]. Historical data indicate this association persists across diverse viral infections. During the SARS-CoV-1 outbreak (2002–2003), type 2 diabetes was an independent predictor of acute complications and mortality [19]. Subsequently, during the 2009 H1N1 influenza pandemic, T2DM patients faced a fourfold higher risk of ICU admission compared to non-diabetic individuals [20]. In COVID-19, diabetic patients not only face a higher risk of developing severe disease but also exhibit significantly increased mortality rates. Multiple studies have further elucidated the role of diabetes in exacerbating viral infections at both mechanistic and clinical levels.

Lin et al. demonstrated through meta-analysis that age, blood glucose levels, lymphocyte counts, and diabetes are independent risk factors for severe COVID-19, with diabetes carrying the highest risk ratio [21]. Du et al. used logistic regression analysis to emphasize that the mechanism behind worsening disease in elderly diabetic patients with concurrent infection may be related to lipid metabolism disorders caused by long-term hyperglycemia [22].

Under the inflammatory storm triggered by COVID-19, this metabolic background makes patients more susceptible to rapid progression to severe or critical illness, significantly increasing mortality risk. Regarding influenza, multiple studies consistently report higher infection-related risks among diabetic patients. Compared to non-diabetic individuals, T2DM patients exhibit significantly higher rates of influenza events requiring medical intervention (1.96% vs. 1.37%, $p < 0.001$). This group also demonstrates higher vaccination and antiviral medication usage rates, indirectly corroborating their recognized high-risk status [23].

Monitoring of hospitalized patients aged 65 years and older revealed a significantly increased risk of influenza-related hospitalization among diabetic patients (RR = 1.57) [24]. Furthermore, during the influenza infection period (6 weeks before and after diagnosis), the risk of pneumonia co-occurrence in diabetic patients surged to 7.4 times the baseline level, with a significantly higher gain score for new-onset pneumonia compared to non-diabetic controls (4.76% vs. 3.06%) [23]. More critically, diabetic patients exhibited markedly elevated risks of ICU admission due to influenza (RR = 1.84), mechanical

ventilation requirement (RR = 1.95), and in-hospital mortality (RR = 1.48), further underscoring diabetes' role in driving influenza severity [24].

In summary, whether during coronavirus or influenza outbreaks, diabetes serves as a significant independent risk factor that substantially increases the risk of severe illness, healthcare demands, and mortality among infected individuals. The underlying mechanisms are closely associated with hyperglycemia-mediated metabolic disorders and immune dysregulation.

4.2. Viral pneumonia leads to glucose metabolic disorders

Viral respiratory infections (such as COVID-19) may exacerbate glucose metabolic disorders through inflammatory responses and hormonal changes. Previous observations of severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) also indicate that inflammatory cell infiltration can impair hepatic insulin signaling, suppress insulin-mediated glucose uptake, and result in hyperinsulinemia and hyperglycemia [25].

Diabetic patients infected with SARS-CoV-2 are more prone to entering a high-stress state, triggering massive release of hyperglycemic hormones (such as glucocorticoids and catecholamines), which in turn causes significant hyperglycemia and abnormal blood glucose variability [26]. Multiple clinical studies provide population-based evidence linking viral pneumonia to glucose metabolism abnormalities.

An investigation of 551 hospitalized COVID-19 patients in Italy found that 46% developed elevated blood glucose levels despite no prior history of diabetes; follow-up revealed that 35% of recovered patients still exhibited persistent hyperglycemia six months later [27]. Another large-scale study further indicated that COVID-19 patients face approximately a 40% increased risk of developing new-onset diabetes one year post-infection compared to healthy individuals [28]. Disease severity positively correlates with diabetes risk: patients requiring hospitalization or intensive care treatment exhibited roughly a threefold elevated risk compared to controls, while even mild cases without hospitalization showed increased susceptibility.

5. Prevention and future outlook

5.1. Vaccination

Vaccination serves as an effective means of preventing viral pneumonia, particularly for high-risk groups prone to complications, such as individuals with diabetes and the elderly. Studies indicate that vaccination reduces the risk of severe influenza cases progressing to pneumonia by approximately 15% [29]. Furthermore, another study on respiratory syncytial virus (RSV) demonstrated that during epidemic seasons, vaccination provided protection against RSV-related hospitalizations and severe in-hospital outcomes [30]. Specifically, efficacy against acute respiratory failure was 73% (95% CI: 50%–86%), while efficacy against invasive mechanical ventilation or death was 72% (95% CI: 7%–91%).

Further analysis of breakthrough infections with the Delta variant (VOC) suggested that prior vaccination significantly mitigated the severity of pneumonia caused by heterologous SARS-CoV-2 infection and reduced the virus-induced inflammatory response [31]. These findings indicate that current vaccination strategies not only help control transmission of known viral strains but may also enhance cross-protection against heterologous SARS-CoV-2 variants, thereby reducing the risk of severe symptoms following infection across the entire population. Consequently, advancing vaccination programs holds positive and far-reaching significance for establishing herd immunity barriers and reducing the burden of severe illness and mortality associated with viral pneumonia.

5.2. Glycemic control

Glycemic control is a critical component of comprehensive treatment for viral pneumonia, helping to reduce infection risk, improve prognosis, minimize complications, and enhance immune responses. Hyperglycemia impairs pulmonary immune function, particularly the antigen-presenting capacity of dendritic cells, leading to weakened immune responses and

increased susceptibility to viral pneumonia [15]. Controlling blood glucose maintains normal function of pulmonary immune cells, thereby lowering infection risk. Patients with well-controlled blood glucose (fluctuating within 3.9–10.0 mmol/L) exhibit significantly lower mortality rates from viral pneumonia.

For instance, studies on COVID-19 patients with diabetes show mortality rates of only 1.1% among those achieving target glycemic control, compared to 11% in those with poor control [32]. Hyperglycemia exacerbates inflammatory responses, leading to multi-organ dysfunction. Effective glycemic control helps mitigate inflammation, reduce damage to the lungs and other organs, and promote recovery. Stable blood glucose prevents suppression of immune cell functions, such as lymphocyte activity and phagocyte function. This enhances the body's ability to recognize and eliminate viruses, shortening the disease course [26,33,34].

5.3. Future outlook

The population of individuals with diabetes complicated by viral pneumonia warrants attention. Future research and management of this condition require establishing a multidimensional prevention and control system spanning molecular mechanisms to public health policies. Epidemiologically, studies must move beyond risk association confirmation to develop dynamic predictive models integrating real-time blood glucose, complications, and immune biomarkers, while elucidating the bidirectional causal relationship between infection and diabetes. Although it is well established that hyperglycemia impairs antigen-presenting function in dendritic cells and causes T-cell cytotoxicity exhaustion, the upstream signaling pathways and cell-specific metabolic mechanisms driving these effects remain unknown.

Further research in this area is needed, with validation in human populations, to address the vulnerability of vulnerable populations—such as individuals with diabetes—to viral infections. Preventive measures should incorporate vaccines and precision glycemic control. Ultimately, interdisciplinary collaboration must accelerate the translation of fundamental discoveries into clinical applications, alongside public health policies ensuring drug and vaccine accessibility. The goal is to significantly reduce related mortality rates and break the vicious cycle linking infection to metabolic disorders.

Disclosure statement

The author declares no conflict of interest.

References

[1] Naghavi M, Ong K, Aali A, et al., 2024, Global Burden of 288 Causes of Death and Life Expectancy Decomposition in 204 Countries and Territories and 811 Subnational Locations, 1990-2021: A Systematic Analysis for the Global Burden of Disease Study 2021. Lancet, 403(10440):2100–2132.

[2] Li Y, Hao E, Liu J, et al., 2021, Research Progress on Pharmacological Effects of Traditional Medicines in China and ASEAN Countries in Treatment of Diabetes and Its Complications. Chinese Traditional and Herbal Drugs, 52(4): 1165–1176.

[3] Ong K, Stafford L, McLaughlin S, et al., 2023. Global, Regional, and National Burden of Diabetes from 1990 to 2021, with Projections of Prevalence to 2050: A Systematic Analysis for the Global Burden of Disease Study 2021. Lancet, 402(10397): 203–234.

[4] Zhou Y, Liu J, Zhao Z, et al., 2025, The National and Provincial Prevalence and Non-Fatal Burdens of Diabetes in China from 2005 to 2023 with Projections of Prevalence to 2050. Mil Med Res, 12(1):28.

[5] Liu J, Liu M, Chai Z, et al., 2023, Projected Rapid Growth in Diabetes Disease Burden and Economic Burden in China: A Spatio-Temporal Study from 2020 to 2030. Lancet Reg Health West Pac, 3(33):100700.

[6] Qu J, Zhou M, 2020, Diagnosis and Treatment Pathway for Pneumonia in Patients with Diabetes Mellitus: A Chinese Experts' Consensus. Chin J Endocrinol Metab, 36(8):635–642.

[7] Apicella M, Campopiano M, Mantuano M, et al., 2020, COVID-19 in People with Diabetes: Understanding the Reasons for Worse Outcomes. Lancet Diabetes Endocrinol, 8(9):782–792.

[8] Shi Q, Zhang X, Jiang F, et al., 2020, Clinical Characteristics and Risk Factors for Mortality of COVID-19 Patients With Diabetes in Wuhan, China: A Two-Center, Retrospective Study. Diabetes Care, 43(7):1382–1391.

[9] Brufsky A, 2020, Hyperglycemia, Hydroxychloroquine, and the COVID-19 Pandemic. J Med Virol, 92(7):770–775.

[10] Means C, 2020, Letter to the Editor: Mechanisms of Increased Morbidity and Mortality of SARS-CoV-2 Infection in Individuals with Diabetes: What This Means for an Effective Management Strategy. Metabolism, 2020(108):154254.

[11] Wang J, Meng W, 2020, COVID-19 and Diabetes: The Contributions of Hyperglycemia. J Mol Cell Biol, 12(12):958–962.

[12] Liu W, Li H, 2020, COVID-19:Attacks the 1-Beta Chain of Hemoglobin and Captures the Porphyrin to Inhibit Human Heme Metabolism. ChemRxiv. https://doi.org/10.26434/chemrxiv.11938173.v9

[13] Codo A, Davanzo G, Monteiro L, et al., 2020, Elevated Glucose Levels Favor SARS-CoV-2 Infection and Monocyte Response Through a HIF-1α/Glycolysis-Dependent Axis. Cell Metab, 32(3):437–446

[14] Herder C, Roden M, Venteclef N, et al., 2024, Diabetes and Pulmonary Infection: How Hyperglycaemia Shapes the Immune System. Signal Transduct Target Ther, 9(1):67.

[15] Gray V, Chen W, Tan R, et al., 2024, Hyperglycemia-Triggered Lipid Peroxidation Destabilizes STAT4 and Impairs Anti-Viral Th1 Responses in Type 2 Diabetes. Cell Metab, 36(12):2511–2527.

[16] Šestan M, Mikašinović S, Benić A, et al., 2024, An IFNγ-Dependent Immune-Endocrine Circuit Lowers Blood Glucose to Potentiate the Innate Antiviral Immune Response. Nat Immunol, 25(6):981–993.

[17] Nobs S, Kolodziejczyk A, Adler L, et al., 2023, Lung Dendritic-Cell Metabolism Underlies Susceptibility to Viral Infection in Diabetes. Nature, 624(7992):645–652.

[18] Singh A, Khunti K, 2022, COVID-19 and Diabetes. Annu Rev Med, 2022(73):129–147.

[19] Yang J, Feng Y, Yuan M, et al., 2006, Plasma Glucose Levels and Diabetes are Independent Predictors for Mortality and Morbidity in Patients with SARS. Diabet Med, 23(6):623–8.

[20] Allard R, Leclerc P, Tremblay C, et al., 2010, Diabetes and the Severity of Pandemic Influenza A (H1N1) Infection. Diabetes Care, 33(7):1491–1493.

[21] Lin X, Zhang C, Kong H, et al., 2022, Analysis on Impacts of Diabetes on the Severity and Mortality of Patients with COVID-19: A Meta-Analysis. Clinical Focus, 37(5):389–399.

[22] Du Y, Guo Y, Jiang L, 2022, Influence of Chronic Complications of Type 2 Diabetes on the Condition of COVID-19 in the Elderly. Chin J Public Health Eng, 21(5):846–847.

[23] Samson S, Konty K, Lee W, et al., 2021, Quantifying the Impact of Influenza Among Persons With Type 2 Diabetes Mellitus: A New Approach to Determine Medical and Physical Activity Impact. J Diabetes Sci Technol, 15(1):44–52.

[24] Owusu D, Rolfes M, Arriola C, et al., 2022, Rates of Severe Influenza-Associated Outcomes Among Older Adults Living With Diabetes-Influenza Hospitalization Surveillance Network (FluSurv-NET), 2012-2017. Open Forum Infect Dis, 9(5):131.

[25] Kazakou P, Lambadiari V, Ikonomidis I, et al., 2022, Diabetes and COVID-19: A Bidirectional Interplay. Front Endocrinol (Lausanne), 2022(13):780663.

[26] Wang A, Zhao W, Xu Z, et al., 2020, Timely Blood Glucose Management for the Outbreak of 2019 Novel Coronavirus Disease (COVID-19) is Urgently Needed. Diabetes Res Clin Pract, 2020(162):108118.

[27] Montefusco L, Ben M, D'Addio F, et al., 2021, Acute and Long-Term Disruption of Glycometabolic Control After SARS-CoV-2 Infection. Nat Metab, 3(6):774–785.

[28] Xie Y, Al-Aly Z, 2022, Risks and Burdens of Incident Diabetes in Long COVID: A Cohort Study. Lancet Diabetes Endocrinol, 10(5): 311–321.

[29] Godoy P, Soldevila N, Martínez A, et al., 2024, Effectiveness of Influenza Vaccination and Early Antiviral Treatment in Reducing Pneumonia Risk in Severe Influenza Cases. Vaccines (Basel), 12(2):173.

[30] Surie D, Self W, Yuengling K, et al., 2025, RSV Vaccine Effectiveness Against Hospitalization Among US Adults Aged 60 Years or Older During 2 Seasons. JAMA, 2025:e2515896.

[31] Fan Q, Shi J, Yang Y, et al., 2022, Clinical Characteristics and Immune Profile Alterations in Vaccinated Individuals with Breakthrough Delta SARS-CoV-2 Infections. Nat Commun, 13(1):3979.

[32] Zhu L, She Z, Cheng X, et al., 2020, Association of Blood Glucose Control and Outcomes in Patients with COVID-19 and Pre-Existing Type 2 Diabetes. Cell Metab, 31(6):1068–1077.

[33] Ceriello A, 2020, Hyperglycemia and COVID-19: What Was Known and What is Really New? Diabetes Res Clin Pract,167:108383.

[34] Ma W, Ran X, 2020, The Management of Blood Glucose Should Be Emphasized in the Treatment of COVID-19. J Sichuan Univ (Med Sci Ed), 51(2):146–150.