

# RSSDI Indian Diabetes

EDUCATOR JOURNAL



**Theme of the Month**

**Diabetes and Respiratory Health**

To keep Members of Diabetes Care team abreast about  
DSME/DSMS - (Diabetes Self management Education/Support) Concepts

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## FOREWORD

Research Society for the Study of Diabetes in India (RSSDI) founded by Prof. MMS Ahuja in the year 1972 is the biggest scientific association of healthcare professionals involved in promoting diabetes education and research in India. RSSDI is happy to collaborate with USV to support their endeavour to make India the 'Diabetes care capital of the world'. Through this collaboration, RSSDI would like to strengthen the cadre of diabetes educators by empowering them with recent updates in diabetes management helping bridge the gap between the physician and the patient. Today, the rule of 50% is prevailing in terms of awareness, detection, treatment and control in T2DM. Our aspiration is to achieve 90-90-90-90 i.e. 90% of people with diabetes should be made aware, 90% should be detected, 90% of those detected should be treated, and 90% of those treated should reach their goals.

Indian Diabetes Educator Journal (IDEJ) is the first of its kind in India, and the longest running monthly diabetes educator journal since April 2015 and continues its endeavour to spread awareness, knowledge and enable healthcare teams to manage individuals with diabetes and empower them for self-care. RSSDI IDEJ will continue to keep the members of diabetes care team abreast with concepts of Diabetes Self-Management Education/Support (DSME/S) with a reach of 44000 doctors and diabetes educators digitally.

Diabetes being a systemic disease, also affects the lungs, causing restrictive types of ventilatory changes probably because of glycosylation of connective tissues, reduced pulmonary elastic recoil, and inflammatory changes in the lungs. The true relationship between pulmonary diseases and diabetes mellitus has not been clarified. This month's issue of IDEJ tries to touch upon the association of diabetes with lung-related comorbidities and strategies to prevent and treat them.

We sincerely thank our contributors for making this issue delightful reading for our readers. We dedicate this journal to all the healthcare professionals who are working relentlessly towards making "India-The Diabetes Care Capital of the World."

Sincere Regards,

Dr. Sanjay Agarwal  
RSSDI Secretary

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# Cover Story: Uncovering the Diabetes and Lung Disease Connection



**Dr. Siva Subrahmanyam**

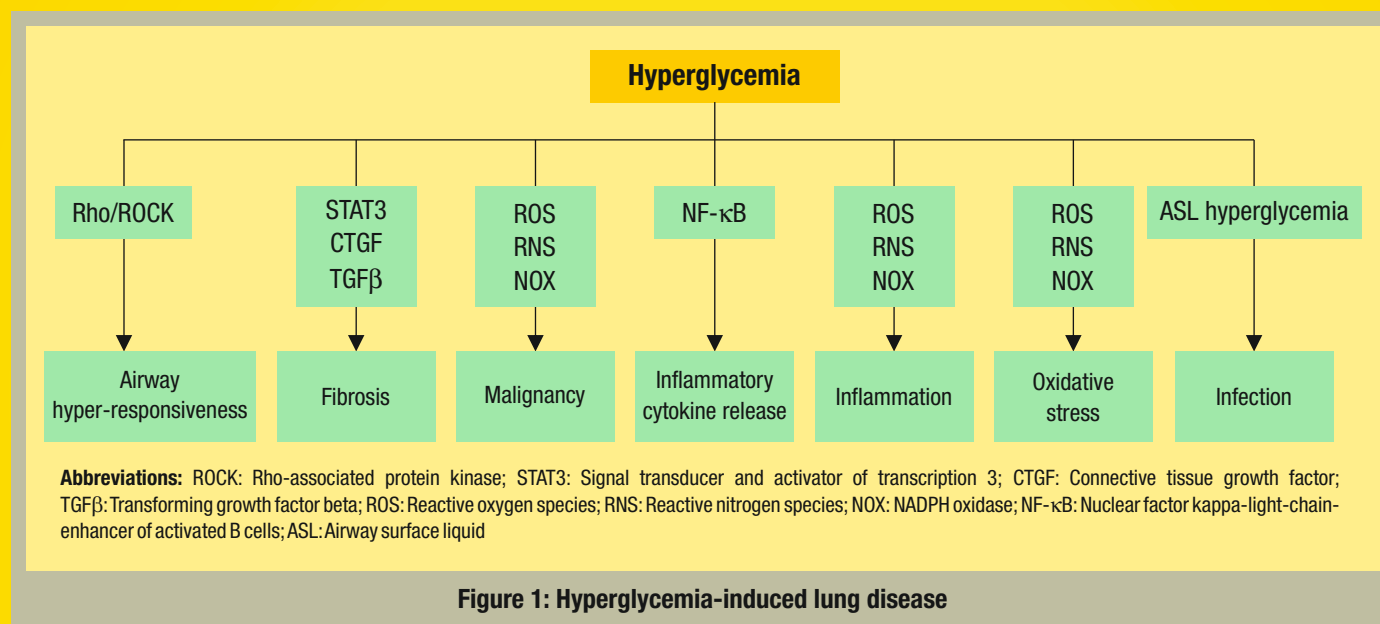
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Diabetes complications, such as micro and macrovascular damage to many organs, especially the kidney, retina, and cardiovascular system, have been widely investigated. Although diabetes has been shown to affect almost every organ in the body, the lung is one of the most neglected target organs. Individuals with diabetes frequently report

respiratory symptoms and are at increased risk of several pulmonary diseases.

The underlying mechanisms for lung dysfunction in individuals with diabetes include hyperglycemia, oxidative stress, hyperinsulinemia, autonomic neuropathy, micro/macroangiopathy of alveolar capillaries and pulmonary arterioles, glycosylation of tissue proteins, collagen and elastin changes, malfunction of respiratory muscles, alteration of connective tissue and surfactant dysfunction. The figure below is a schematic representation of hyperglycemia-induced lung disease.



Hyperglycemia, through various pathways, contributes to airway hyper-responsiveness, accelerates lung fibrosis, and enhances cancer cell growth, chronic inflammation, inflammatory cytokine release, and oxidative stress. Additionally, higher glucose concentrations in airway surface liquid (ASL) and lung immune cell dysfunction due to hyperglycemia increase susceptibility to pulmonary infections. This article discusses commonly prevalent lung disease and its association with diabetes.



**Asthma:** The risk of asthma in individuals with diabetes is over twice that of individuals without diabetes (hazard ratio [HR] of 2.2). Diabetes in asthma patients is said to cause more severe asthma symptoms, such as higher exacerbation rates causing frequent emergency department visits. It has also been shown to induce sputum hypersecretion and impact long-term mortality.

**Chronic obstructive pulmonary disease (COPD):** COPD is associated with a chronic systemic inflammatory condition. The continuously elevated levels of inflammatory mediators, reflecting the enhanced inflammatory state seen in COPD, may contribute to the development of diabetes. Prevalence estimates of diabetes among COPD patients range between 10% and 23%. The risk of diabetes in COPD patients is higher in more severe phenotypes (levels 3 and 4 according to the Global Initiative for Chronic Obstructive Lung Disease [GOLD] guidelines). This risk is independent of body mass index (BMI), smoking, and other confounding factors. Additionally, the presence of diabetes in individuals with COPD is associated with worse outcomes, including increased mortality and hospitalization rates.

**Idiopathic pulmonary fibrosis (IPF):** Many comorbid conditions, such as coronary artery disease, pulmonary hypertension, gastroesophageal reflux, and diabetes, are known to occur in IPF. IPF patients with diabetes are shown to have higher incidences of hypertension, cardiovascular diseases, and other malignancies compared to those without diabetes.

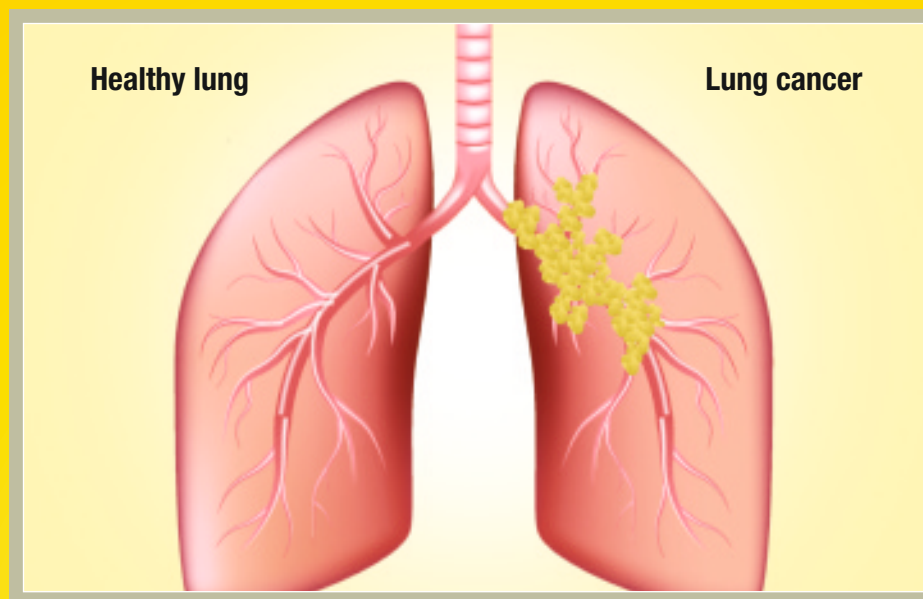
**Pulmonary hypertension:** Diabetes and pulmonary hypertension are strongly associated. Diabetes-related microvascular and macrovascular damage may affect pulmonary vasculature and increase its susceptibility to the development and progression of pulmonary hypertension and may play a role in patient prognosis and survival.





**Lung cancer:** Diabetes is said to influence lung cancer progression and outcome and may serve as a poor prognostic factor for lung cancer. Proper glycemic control for lung cancer patients is required to induce antineoplastic effects and increase survival.

Thus, emerging evidence suggests that diabetes may affect the pathogenesis, development, and progression of several lung diseases, their prognosis, and clinical outcomes. Diabetes should be regarded as a significant factor in the clinical management of patients with lung disease. The pro-inflammatory, proliferative, and oxidative effects of hyperglycemia play a crucial role in impacting the pulmonary vasculature, airways, and lung parenchyma.



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# Tuberculosis and Diabetes: Two-way Connection



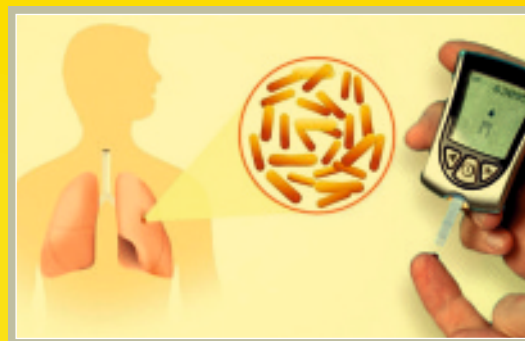
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India has the highest number of tuberculosis (TB) cases in the world, with an estimated 2.8 million cases per year and an incidence rate of 217 per 1,00,000 as of 2015. According to the International Diabetes Foundation (IDF) Diabetes Atlas (2021), 10.5% of the adult population (20–79 years) in the world have diabetes mellitus (DM). India accounts

for 1 in 7 of all adults living with diabetes worldwide. The significant prevalence of DM and TB in epidemic proportions has correctly earned them the labels “the converging epidemics” and “double burden.”

Since 1000 A.D., reports have linked DM and TB. Greek scientist Avicenna observed that diabetes increased the likelihood of TB, noting the frequent complication of “phthisis” (Greek for TB) in people with DM. TB and diabetes have common risk factors, including poverty, malnutrition, and urbanization. These factors lead to the epidemiological overlap of both diseases, especially in developing nations like India, where TB and diabetes are highly prevalent. Recent research has confirmed a significant two-way connection between TB and DM.



## DM as a risk factor for TB



Diabetes is on the rise globally, particularly in India, where TB is already endemic. According to the World Health Organization (WHO), diabetes is linked to 15% of TB cases worldwide. Of this 15%, over 40% of the cases are in India. Immune deficiency can reactivate latent TB. TB prevalence is well-known in diseases like human immunodeficiency virus (HIV), but DM is a more significant risk factor due to its wider prevalence. Studies show higher multidrug-resistant TB in people with DM (odds ratio of 2.1), indicating immune dysregulation. The immune response in DM is hyper-reactive yet ineffective, potentially damaging pulmonary tissue.

Chronic hyperglycemia impairs both innate and adaptive immunity. DM weakens cell-mediated immunity, affects cytokine response, and alters alveolar macrophage defenses. It disrupts neutrophil recruitment, monocyte movement, and macrophage phagocytosis. T-helper cell activation and interferon-gamma release are ineffective. Altered pulmonary microvasculature and micronutrient deficiencies further aid TB invasion. This chronic immunosuppression predisposes an individual with DM to a higher risk and bacilli load of TB infection.

## TB as a risk factor for DM

Studies have shown a high prevalence of diabetes, as well as impaired glucose tolerance, in patients with TB. DM may affect up to 35% of TB patients, yet published statistics on this topic are inconsistent. In most of these cases, the impaired glucose tolerance reverts back to normal after successful treatment for TB. However, the increased risk of developing DM persists. Chronic inflammation and metabolic dysregulation associated with TB infection may contribute to the development of insulin resistance and subsequent diabetes.



According to WHO, the prevalence of DM is negatively associated with both TB incidence and mortality, doubling to tripling the risk of developing TB, doubling the risk of death during TB treatment, quadrupling the risk of TB relapse after treatment, and doubling the risk of multidrug-resistant TB (MDR-TB).

The confluence of TB and DM poses considerable challenges for public health systems, especially in low- and middle-income countries where both diseases are common. A substantial proportion of people with coexisting diabetes and TB remain undiagnosed or are diagnosed at a late stage. Lack of early detection and treatment gives rise to complications from TB-diabetes comorbidity, which leads to an increased cost of treatment and out-of-pocket expenditure. Early detection aids in improving the management of both diseases. Hence, the synergism involving DM and TB demands bidirectional screening. Screening tests include sputum microscopic examination, rapid molecular testing, random plasma glucose, and glycosylated hemoglobin, each with advantages and disadvantages of its own.

Effective treatment of both TB and DM, in conjunction with aggressive DM screening among the TB population, could improve individual outcomes. Preventive measures such as TB vaccination and rigorous DM prevention and management are more effective ways to make a difference at the community level.

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# Interstitial Lung Disease and Pulmonary Fibrosis in Diabetes



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Diabetes mellitus (DM) is a metabolic disorder marked by insulin deficiency or resistance, chronic hyperglycemia, and damage to the micro and macrovascular tissues. The lung has a dense alveolar-capillary network and connective tissue, implying that diabetic microvascular injury could target it.

Several studies in recent years have shown that hyperglycemia can cause lung disorders such as interstitial fibrotic alterations, alveolar microangiopathy, and idiopathic pulmonary fibrosis (IPF).

## Interstitial lung disease (ILD) and diabetes

ILD, also known as diffuse parenchymal lung disease, refers to a diverse group of lung ailments that are grouped based on common clinical, radiological, physiologic, or pathologic criteria. The pathophysiology of DM, which causes ILD, is multifaceted and complex. Hyperglycemia induces oxidative stress and an imbalance between free radical formation and antioxidant activity, which contributes to lung dysfunction in DM. DM frequently leads to autonomic neuropathy, which can influence the pulmonary vascular tone, leading to pulmonary hypertension and phrenic nerve neuropathy, which can result in diaphragmatic dysfunction. These individuals often present with unexplained dyspnea and orthopnea. Autonomic neuropathy also impairs pulmonary mechanoreceptors, resulting in decreased airway smooth muscle tone and excessive surfactant production. Long-term hyperglycemia causes non-enzymatic glycation of extracellular proteins in the pulmonary interstitium, which contributes to ILD in DM. DM-induced microangiopathy of pulmonary capillaries, along with glycosylation of alveolar basement membrane proteins, severely affects alveolar gas exchange.





## IPF and diabetes

IPF is a chronic, progressive, fibrotic ILD that has the histologic appearance of typical interstitial pneumonia. The actual pathophysiology of IPF is not entirely understood. Traditionally, the progression of IPF was separated into three stages: (1) Initiation stage, oxidative stress damage from all causes is the initiation factor of IPF; (2) Progression stage, inflammation of alveoli, activation of immune cells, and secretion of various pro-inflammatory factors, causing injuries of epithelial, endothelial, and interstitial cells, collagenous tissues, and basement membranes; (3) Outcome stage, the production of pulmonary fibrosis, characterized by proliferation of fibroblasts and myofibroblasts, deposition of extracellular matrix (ECM), and degradation of structure in lung tissues, finally leading to chronic respiratory failure. Pathological pulmonary fibrosis is characterized by epithelial-mesenchymal transition (EMT), which increases fibrotic markers like  $\alpha$ -smooth muscle actin ( $\alpha$ -SMA) and vimentin while decreasing epithelial markers like E-cadherin.

Diabetes, IPF, and diabetic microangiopathy are becoming more common each year. Early detection and monitoring of invasive pneumococcal disease (IPD) and ILD may minimize pulmonary comorbidities.



### Resources:

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# Coronavirus Disease 2019 (COVID-19) and its Association with Diabetes



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COVID-19 has been found to have a significant association with diabetes mellitus. Studies have shown that patients who have had severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection leading to COVID-19 may manifest not only with new-onset diabetes but also with worsening of pre-existing diabetes.

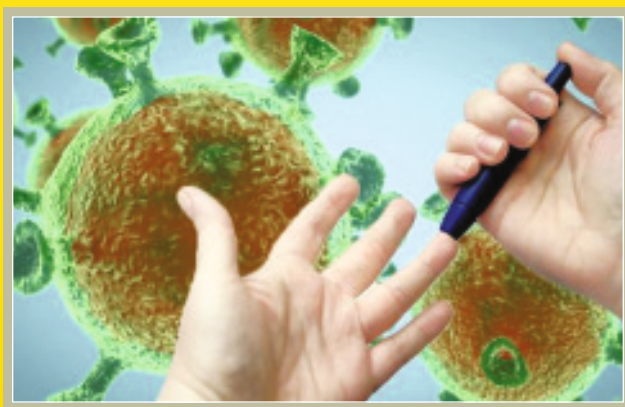
The resulting hyperglycemia is known to influence the clinical outcome, increasing morbidity and mortality in some cases.

Several mechanisms have been proposed to explain this association.

1. Cytopathic effect and autoimmune destruction of insulin-secreting pancreatic beta cells: The virus may directly damage these cells, leading to impaired insulin secretion.
2. Cytokine storm during the active phase of infection: This can cause impaired insulin secretion and resistance, contributing to hyperglycemia.
3. Drug-induced hyperglycemia: Certain medications used to treat COVID-19, such as glucocorticoids, can increase blood glucose levels.
4. Undetected pre-existing hyperglycemia: Some patients may have undiagnosed diabetes that is revealed during the course of their COVID-19 illness.
5. Stress-induced impairment of glucose metabolism: The stress of the infection can disrupt glucose metabolism, leading to hyperglycemia.

According to a meta-analysis and systematic review, survivors of COVID-19 had a 66% increased risk of developing diabetes. Another study noted that the presence of diabetes could be considered a predictor for the progression and outcomes of COVID-19, with potential mechanisms including overactivated inflammation and imbalanced immune responses.

Overall, the correlation between diabetes and COVID-19 highlights the importance of active monitoring of glucose dysregulation after recovery from SARS-CoV-2 infection.



## Key points

- COVID-19 is linked to new-onset and worsening diabetes, increasing morbidity and mortality due to hyperglycemia.
- The mechanisms include direct damage to pancreatic beta cells, cytokine storms impairing insulin, medication-induced hyperglycemia, undiagnosed diabetes, and stress-induced glucose metabolism disruption.
- A study found a 66% higher risk of diabetes in COVID-19 survivors, with diabetes predicting worse outcomes due to inflammation and immune imbalances.
- Active glucose monitoring post-COVID-19 is essential to manage potential long-term health impacts.



## Resources:

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3. Ssentongo P, Zhang Y, Witmer L, Chinchilli VM, Ba DM. Association of COVID-19 with diabetes: A systematic review and meta-analysis. *Sci Rep*. 2022;12(1):20191. Published 2022 Nov 23. doi:10.1038/s41598-022-24185-7

## Interview with Dr. Dharmendra V. Dubey



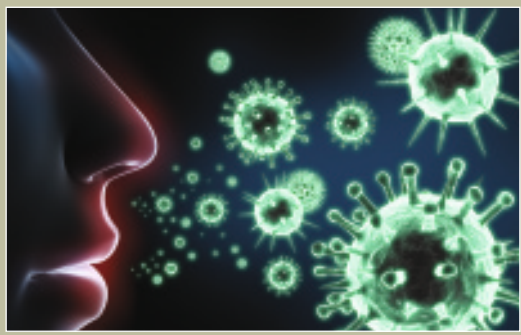
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Dr. Dharmendra Dubey is committed to providing comprehensive care for his patients, leveraging his extensive training and experience to address various respiratory conditions effectively.

### Diabetes and Respiratory Health



**1. What are the causes of respiratory diseases among individuals with diabetes?**

**Ans.** Respiratory diseases among individuals with diabetes are mainly due to the increased susceptibility of patients to various infective agents and also poor response to treatment in view of the hyperglycemic state.

**2. Which respiratory or lung diseases as comorbidities do you commonly see in your practice in individuals with diabetes?**

**Ans.** Obstructive sleep apnea (OSA) and chronic obstructive pulmonary disease (COPD) are commonly seen as comorbidities in individuals with diabetes. Obesity is a common predisposing factor for both diabetes mellitus and OSA. Repeated lung infections in individuals with diabetes lead to poor lung function over time with the development of COPD.



**3. Are there specific challenges or considerations in managing respiratory conditions in individuals with diabetes compared to those without diabetes?**

**Ans.** The special challenge in managing respiratory conditions in individuals with diabetes is that the hyperglycemic state needs to be under strict control when a patient is on treatment for an infectious disease. Patients with cardiovascular disease and those on medications like thiazolidinediones are predisposed to heart failure and increased chances of pulmonary edema.



**4. What is your opinion on the use of yoga practices, such as pranayama, in the management of respiratory diseases and diabetes?**

**Ans.** Pranayama, related to respiratory and chest wall muscles, helps maintain lung function and also has some improvement in lung function. Also, it helps to maintain the elasticity and static recoil of the chest wall, which is very useful in restrictive lung diseases. Also, pranayama and yoga help maintain the hormonal balance in the body, improve blood flow to vital organs, lower blood sugar levels, and increase

the utilization of glucose by cells of the body. It helps to achieve mental well-being wherein the stress levels are under control, which is very common these days due to hectic work life, and this helps to prevent the further worsening of diabetes control.

**5. What preventive measures or lifestyle interventions do you recommend for patients with diabetes to maintain optimal respiratory health?**

**Ans.** To maintain optimal respiratory health in individuals with diabetes mellitus, the following things need to be followed.

- a) Strict blood sugar control
- b) Regular 20–30 minutes physical exercise
- c) Yoga and pranayama
- d) Vaccination of individuals with diabetes against influenza and pneumococcal infections
- e) Adequate hydration of the body





# Diabetes and the Risk of Lung Cancer



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Lung cancer, one of the leading causes of cancer-related deaths, involves uncontrolled cell growth in the lungs. Studies have shown that hyperglycemia encourages lung cancer growth. Furthermore, elevated insulin receptor expression is associated with lung cancer progression, and the insulin-like growth factor-1 (IGF-1) and insulin-like growth factor-1 receptor (IGF-1R) pathway play a crucial role in the development of lung cancer.

## The link between diabetes mellitus and lung cancer

A study has shown that type 2 diabetes mellitus (T2DM) not only affects the clinical outcomes of chronic obstructive pulmonary disease (COPD) but also raises the risk of lung cancer in COPD patients. Research suggests that diabetes-induced microangiopathy can impact the lungs, causing histological changes, functional abnormalities, and chronic inflammation at the molecular level.

Multiple case-control studies have demonstrated that T2DM significantly reduces pulmonary function compared to healthy individuals. Hyperglycemia has been observed to increase the levels of inflammatory cytokines, such as interleukin-6 and tumor necrosis factor-alpha, which are crucial in epithelial-to-mesenchymal transition and tumor development.

In one study, patients with both COPD and T2DM had higher white blood cell counts, lower hemoglobin levels, and higher mean C-reactive protein levels compared to those with COPD alone. These findings indicate that T2DM exacerbates systemic inflammation in patients with COPD.

In conclusion, patients with both COPD and T2DM should be closely monitored for lung cancer, especially smokers and rural residents. This underscores the need for improved healthcare access and smoking cessation programs to reduce lung cancer risk in these vulnerable populations.





## Key points

- T2DM worsens COPD outcomes and increases lung cancer risk in these patients.
- COPD patients with T2DM are shown to have higher white blood cell counts and C-reactive protein levels.
- Diabetes-induced microangiopathy leads to lung changes and chronic inflammation.
- Hyperglycemia increases cytokines like interleukin-6, promoting tumor development.
- T2DM may increase lung cancer risk due to chronic inflammation and hyperglycemia.



## Resources:

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2. Leiter A, Charokopos A, Bailey S, *et al*. Assessing the association of diabetes with lung cancer risk. *Transl Lung Cancer Res*. 2021;10(11):4200-4208. doi:10.21037/tlcr-21-601

In T2DM Uncontrolled on DPP4i + Metformin, SGLT2i + Metformin, SGLT2i + DPP4i

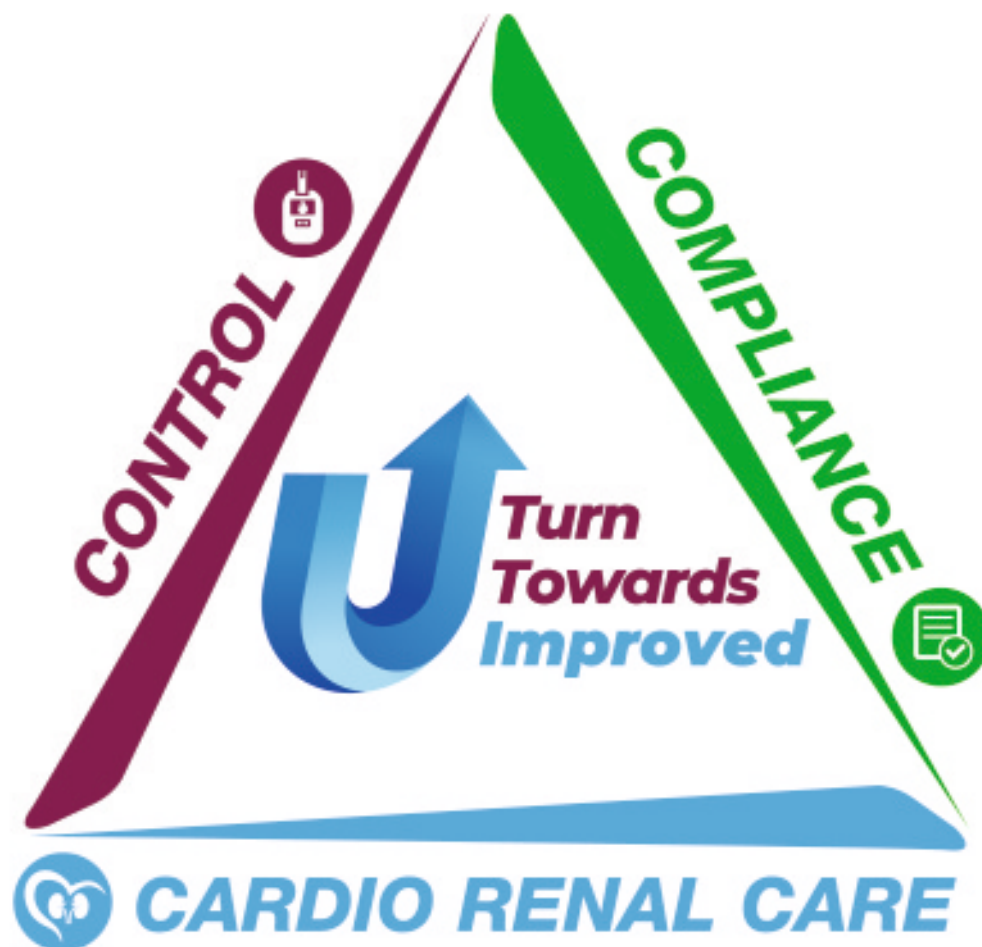
Upgrade with

**UDAPA-Trio**

Dapagliflozin 10 MG + Sitagliptin 100 MG + Metformin 500 MG XR

**UDAPA-Trio Forte**

Dapagliflozin 10 MG + Sitagliptin 100 MG + Metformin 1000 MG XR



#### Abridged Prescribing Information

UDAPA-TRIO Forte, UDAPA-TRIO, Dapagliflozin, Sitagliptin & Metformin Hydrochloride Extended Release Tablets **Composition:** Dapagliflozin 10 mg, Sitagliptin 100 mg & Metformin Hydrochloride Extended Release 1000 mg tablets Dapagliflozin propanediol monohydrate eq. To Dapagliflozin 10 mg Sitagliptin Phosphate Monohydrate IP Eq. Sitagliptin 100 mg Metformin Hydrochloride IP (as Extended Release) 1000 mg Dapagliflozin 10 mg, Sitagliptin 100 mg & Metformin Hydrochloride Extended Release 1000 mg tablets Dapagliflozin propanediol monohydrate eq. To Dapagliflozin 10 mg Sitagliptin Phosphate Monohydrate IP Eq. Sitagliptin 100 mg Metformin Hydrochloride IP (as Extended Release) 500 mg **Indication:** It is indicated as an adjunct to diet and exercise to improve Glycemic Control adults with type 2 diabetes mellitus **Recommended Dosage:** As directed by the physician. **Method of Administration:** Oral **Adverse Reactions:** Most common adverse reactions reported are: Dapagliflozin - Female genital mycotic infections, Nasopharyngitis, Urinary tract infections. Sitagliptin - Upper respiratory tract infection, nasopharyngitis and headache. Metformin - Diarrhea, nausea/vomiting, flatulence, asthenia, indigestion, abdominal discomfort, and headache. **Warnings and Precautions:** Dapagliflozin: Volume depletion; Ketoacidosis in patients with Diabetes Mellitus; Urinary tract infections; Hypoglycemia; Genital mycotic infections Sitagliptin: **General:** Sitagliptin should not be used in patients with type 1 diabetes or for the treatment of Diabetic Ketoacidosis. Acute pancreatitis: Hypoglycemia is used in combinations when combined with other anti-hyperglycemic medicinal product; Renal impairment: Hypersensitivity reactions including anaphylaxis, angioedema, and exfoliative skin conditions - Steven Johnson syndrome; Bullous pemphigoid Metformin Hydrochloride: Lactic acidosis; In case of dehydration (severe diarrhea or vomiting, fever or reduced fluid intake), metformin should be temporarily discontinued and contact with a healthcare professional is recommended. **Contraindications:** Hypersensitivity to the active substance of Dapagliflozin, Sitagliptin & Metformin or to any of the excipients listed. Any type of acute metabolic acidosis (such as lactic acidosis, diabetic ketoacidosis), Diabetic pre-coma; Severe renal failure (eGFR < 30ml/min); Acute conditions with the potential to alter renal function such as: Dehydration, Severe infection, Shock; Acute or chronic disease which may cause those hypoxia such as: Cardiac or respiratory failure, Recent myocardial infarction, Shock, Renal impairment, Acute intoxication, Alcoholism. **Use in special populations:** Pregnant women: Due to lack of human data, drugs should not be used during pregnancy. **Lactating women:** It should not be used during breastfeeding. Pediatric patients: The safety and efficacy of drugs has not yet been established. No data is available. **Geriatric Patients:** In patients >65 years, it should be used with caution as age increases. For Additional Information/full prescribing information, please write to us: USV Private Limited, Arvind Vithal Gandhi Chowk, B.S.D Marg, Govandi, Mumbai - 400088 Last updated on 02/04/2024.



USV Private Limited



Arvind Vithal Gandhi Chowk, B. S. D. Marg, Govandi, Mumbai-400 088. | Tel: 91-22-2556 4048 | Fax: 91-22-2558 4825 | www.usvindia.com

## Guidance on Managing Glycemic Variability: A Doctor's Experience on the MyCare Patient Support Program



**Dr. Pravishal D. Adling**

MBBS, D. Diabetology, PGDCED (RCP-UK)  
Consultant Diabetologist

A 46-year-old man with uncontrolled type 2 diabetes was managed by Dr. Pravishal Adling.

### Here's what Dr. Pravishal Adling has to say:

The patient had uncontrolled diabetes for 5–6 years and presented with a glycated hemoglobin (HbA1c) of 14%. His random sugar was 585 mg/dL. He was a distributor of medicines and stayed with his mother. I started him on premix insulin twice a day. After one week, during his follow-up visit, we noticed that his blood sugar levels were fluctuating. Then we referred him to my MyCare Diabetes Educator (MDE), Ms. Mubarra Taj Mohammed, for counseling. The MDE took a detailed understanding of his situation and realized that he did not have fixed meal timings and at times, he skipped meals even after taking the insulin dose, which led to episodes of hypoglycemia and then, out of hunger, he was eating the wrong foods, leading to hyperglycemia. The MDE explained to him how the insulin works and made him understand why he was having such fluctuations in blood sugar. She advised him to have a fixed routine for meals and to include exercise as part of his daily regime. She gave him a proper understanding of insulin injection techniques, storage of insulin, etc., and made him aware of portion control and the benefits of adding fiber and protein foods to his meals. She also counseled him on the symptoms of hypoglycemia and how to prevent it, and how to correct it with fast carbohydrates in limited portions. He was surprised to know about so many different aspects of blood sugar management. He started adhering to the advice, and slowly, his sugar control is improving, with an HbA1c of 9.3% and fasting glucose of 87 mg/dL.



**Ms. Mubarra Taj Mohammed**

NDEP and T1DE Certified Diabetes Educator

### Here's what the MDE Mubarra Taj Mohammed has to say:

Counseling about insulin, its safety, and benefits, along with regular teleconnects to follow up and counsel on different aspects of diabetes self-management, helped him make sustainable and practical changes in his routine, which led to better blood sugar control.



**MyCARE**  
With me, every step of the way





# MyCARE

With me, every step of the way

20 weeks personalised and hand-holding support for people with diabetes initiated with Insulin.  
Aims to empower PWD\* with information and knowledge they need to ensure a better quality of life while managing their diabetes.



MyCARE Service available at Ahmedabad, Bangalore, Bhopal, Bhuvaneshwar, Burdwan, Chandigarh, Chennai, Cochin, Coimbatore, Delhi, Guwahati, Hubli, Hyderabad, Jaipur, Jodhpur, Kolkata, Lucknow, Ludhiana, Madurai, Meerut, Mumbai, Mysore, Nagpur, Patna, Pune, Siliguri, Surat, Thiruvananthapuram, Varanasi, Vijayawada, Visakhapatnam  
\*PWD: People with Diabetes

In **uncontrolled** T2DM with or at risk of **CVD, HF and CKD**<sup>1</sup>



# UDAPA-S

Dapagliflozin 10 mg + Sitagliptin 100 mg Tablets



turn to a life 'IN RANGE'



Extensively Studied & Recognized Brand of  
**Dapagliflozin + Sitagliptin** in India<sup>#</sup>

Nearly  
**4000**

Patient Data

Real  
World  
Evidence

From  
**550+**

Clinicians Across India

CVD- Cardiovascular Disease HF- Heart Failure CKD- Chronic Kidney Disease

<sup>1</sup>L. Ravikumar et al., Cardiol Cardiovasc Med 2023; 7: 141-144. n= no. of patients [ \*As per the literature search (PubMed and Google Scholar) on 27<sup>th</sup> March 2024

Reference: I. Singh AK, et al., Endocr Pract. 2023 Jul;29(7):509-516.

UDAPA-S

Dapagliflozin and Sitagliptin Tablets

**Composition:** Each Film Coated Tablet Contains: Dapagliflozin Propanediol Monohydrate eq. to Dapagliflozin (10 mg) + Sitagliptin Phosphate Monohydrate IP eq. to Sitagliptin (100 mg)

**Indications:** For the treatment of type 2 diabetes mellitus inadequately controlled on Metformin monotherapy.

**Recommended Dosage:** As directed by the physician.

**Method of Administration:** Oral

**Adverse Reactions:** Female genital mycotic infections, nasopharyngitis, and urinary tract infections are most common adverse reactions associated with dapagliflozin. While, upper respiratory tract infection, nasopharyngitis, and headache are most common adverse reactions associated with sitagliptin.

**Warnings and Precautions: Risk of Volume Depletion in Elderly** - Before initiating Dapagliflozin and Sitagliptin, assess volume status and renal function in the elderly, patients with renal impairment or low systolic blood pressure, and in patients on diuretics. Monitor for signs and symptoms during therapy. **Ketoacidosis in Patients with Diabetes Mellitus** - Assess patients who present with signs and symptoms of metabolic acidosis for ketoacidosis regardless of blood glucose level. If suspected, discontinue UDAPA-S, evaluate and treat promptly. Before initiating UDAPA-S, consider risk factors for ketoacidosis. Patients on UDAPA-S may require monitoring and temporary discontinuation of therapy in clinical situations known to predispose to ketoacidosis. **Urosepsis and Pyelonephritis** - Evaluate for signs and symptoms of urinary tract infections and treat promptly, if indicated. **Hypoglycemia** - Consider a lower dose of insulin or the insulin secretagogue to reduce the risk of hypoglycemia when used in combination with Dapagliflozin and Sitagliptin. **Necrotizing Fasciitis of the Perineum** - Serious, life-threatening cases have occurred in patients with diabetes, both females and males. Assess patients presenting with pain or tenderness, erythema, or swelling in the genital or perineal area, along with fever or malaise. If suspected, institute prompt treatment. **Genital Mycotic Infections** - Monitor and treat if indicated.

**Contraindications:** Patients with a history of hypersensitivity reaction to the active substance or to any of the excipients. In patients with varying degrees of renal impairment, adjusting the dosage is advised based on the severity of the condition. Prohibited medications include strong CYP2C8 inhibitors/inducers, drugs increasing/decreasing hypoglycemic action, drugs known to cause QT prolongation, or other oral hypoglycemic agents other than study medications.

Updated on 20<sup>th</sup> March 2024

For Additional Information/Full prescribing information, please write to us:



USV Private Limited

Arvind Vithal Gandhi Chowk, B.S.D Marg, Govandi, Mumbai-400088



# Coexistence of Type 2 Diabetes Mellitus (T2DM) and Chronic Obstructive Pulmonary Disease (COPD)



**Dr. Pankaj M. Patel**

**MD (Medicine), DNB (Endocrinology)**  
Consultant Endocrinologist and Diabetologist,  
Apex Endocrine-Diabetes Hospital, Rajkot

There is growing evidence that COPD and T2DM frequently coexist, with T2DM being linked to a poorer prognosis. The relationships between these two diseases are complex, multifaceted, and not well understood, but may have significant implications for treatment strategies.

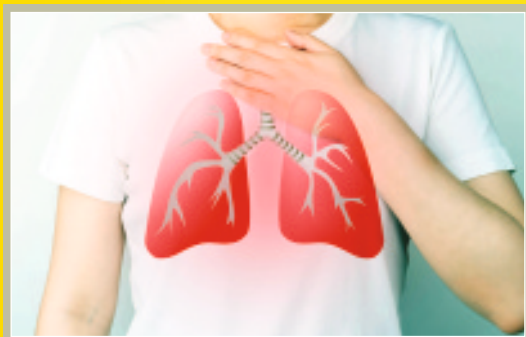
Several studies have indicated a higher incidence of T2DM among COPD patients and vice versa. The co-occurrence of T2DM and COPD is found to be more common in women than in men and in young than in old people, irrespective of gender.

## T2DM in patients with COPD

People with frequent acute exacerbations of COPD (AECOPD) and cardiac comorbidities are more likely to develop T2DM. Frequent exacerbators ( $\geq 2$  treated exacerbations per year) and those on high-dose inhaled corticosteroids (ICS) have a higher risk of developing T2DM. Each 1 mmol/L (18 mg/dL) increase in blood glucose raises the absolute risk of adverse outcomes in AECOPD patients by 15%.



## COPD in patients with T2DM

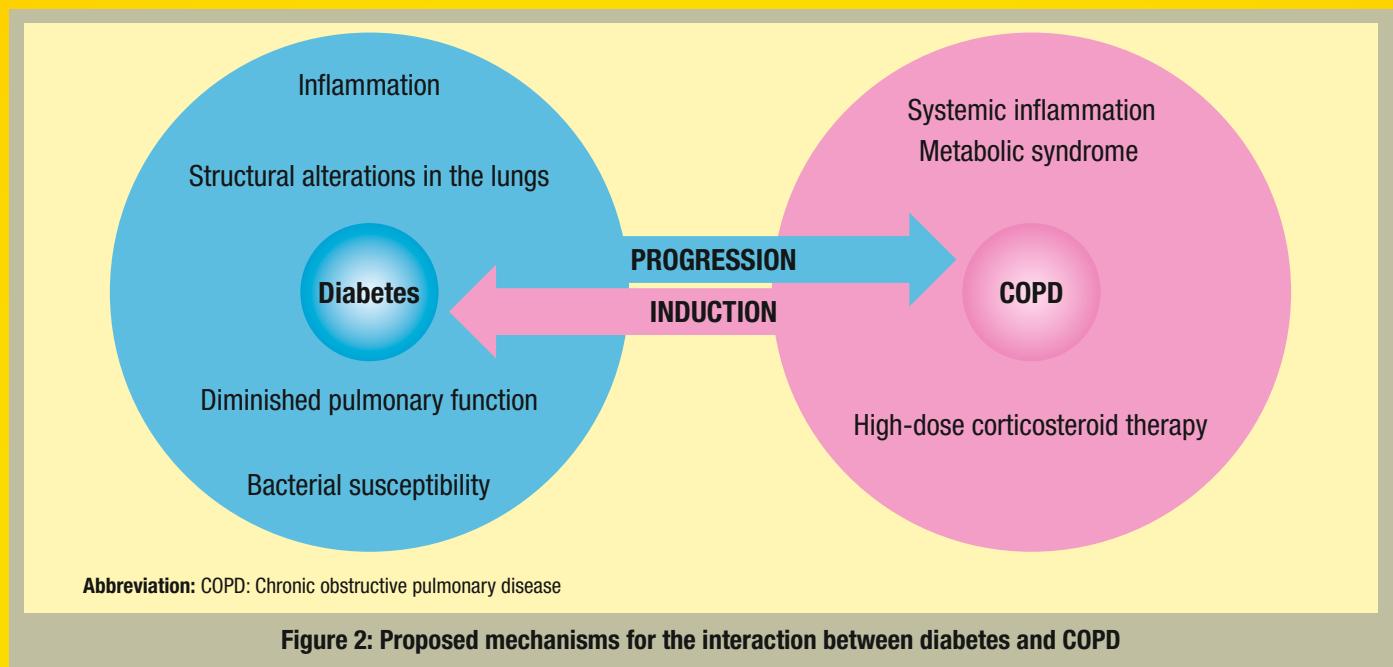


T2DM is associated with a decline in lung function, which may start even before diabetes is diagnosed. Individuals with diabetes mellitus (DM) are shown to have significantly lower forced vital capacity (FVC), forced expiratory volume in 1 second (FEV<sub>1</sub>), mean expiratory flow, expiratory residual volume, total lung capacity, and diffusing capacity for carbon monoxide as compared to people without diabetes. In addition, hyperglycemia also increases mucus production in the airways. Elevated blood glucose levels and HbA1c levels, with or without DM, are found to be strongly associated with an increased risk of subsequent severe AECOPD.

This association remains significant even after adjusting for potential confounders, including age, sex, body mass index (BMI), smoking status, COPD duration, hospitalization frequency for AECOPD, stage of COPD, COPD assessment test (CAT) score, corticosteroid use, hypertension, and cardiovascular disease. Hyperglycemia also increases susceptibility to bacterial infection, further increasing the prevalence of severe AECOPD. Patients with T2DM have a higher risk of hospitalization during AECOPD.

## Potential mechanisms linking COPD and T2DM

Several mechanisms explain the relationship between COPD and diabetes (as shown in the figure below). The core components of T2DM and COPD coexistence are oxidative stress and systemic inflammation. Diabetes may worsen the progression and prognosis of COPD via the consequences of hyperglycemia on lung physiology, inflammation, and susceptibility to bacterial infection. On the other hand, inflammatory processes in COPD and/or the therapeutic side effects related to the use of corticosteroids may increase the risk of developing T2DM.



## Pharmacological interferences

The treatment of COPD and T2DM involves careful consideration of drug interactions. ICS, commonly used in COPD management, can increase blood glucose levels and affect glycemic control. Conversely, some antidiabetic drugs, such as glucagon-like peptide-1 (GLP-1) agonists and sodium-glucose cotransporter-2 (SGLT-2) inhibitors, are found to be associated with a comparatively reduced risk of AECOPD compared to others. Hence, employing shared therapeutic strategies for both conditions could be a practical approach.

Understanding the mechanisms linking COPD and T2DM is crucial for developing effective treatment strategies. Managing systemic inflammation and oxidative stress appears to be a promising approach. Clinicians must consider the potential risks and benefits of pharmacological treatments for both diseases to optimize patient outcomes.

### Resources:

1. Cazzola M, Rogliani P, Ora J, Calzetta L, Lauro D, Matera MG. Hyperglycaemia and Chronic Obstructive Pulmonary Disease. *Diagnostics (Basel)*. 2023;13(21):3362. Published 2023 Nov 1. doi:10.3390/diagnostics13213362.
2. Gläser S, Krüger S, Merkel M, Bramlage P, Herth FJ. Chronic obstructive pulmonary disease and diabetes mellitus: A systematic review of the literature. *Respiration*. 2015;89(3):253-264. doi:10.1159/000369863.



# Diabetes and Obstructive Sleep Apnea



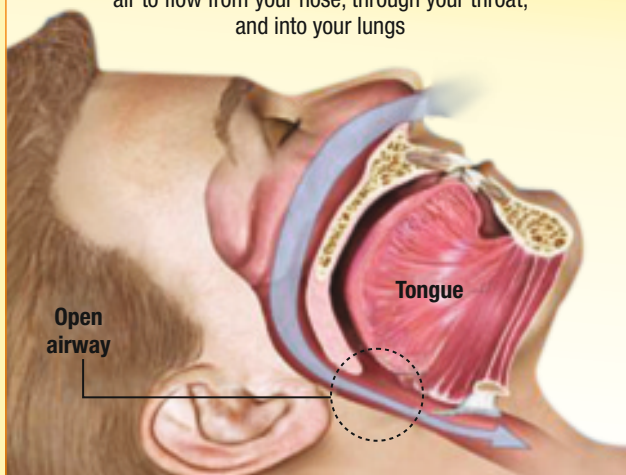
## Dr. M. Surendra Nehru

**MBBS, MD (General Medicine)**  
Senior Consultant Physician,  
Surendra Nehru Hospitals, Telangana

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder in which the relaxation of supportive structures of the throat during sleep leads to the collapse of the retropharyngeal soft tissue, blocking the upper airway. There is habitual snoring, breathing interruption leading to hypoxia (low oxygen levels), awakening with choking or gasping, insomnia, and daytime fatigue with sleepiness.

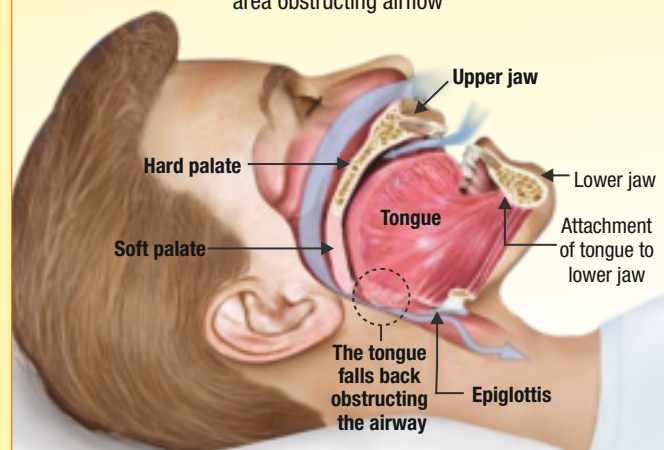
### Normal anatomy and function

Your upper airway is open and unobstructed, allowing air to flow from your nose, through your throat, and into your lungs



### Obstructive sleep apnea

During sleep, gravity and muscle relaxation allow the tongue and surrounding soft tissues to fall back into the throat area obstructing airflow



**Figure 3: Normal anatomy versus obstructive sleep apnea**

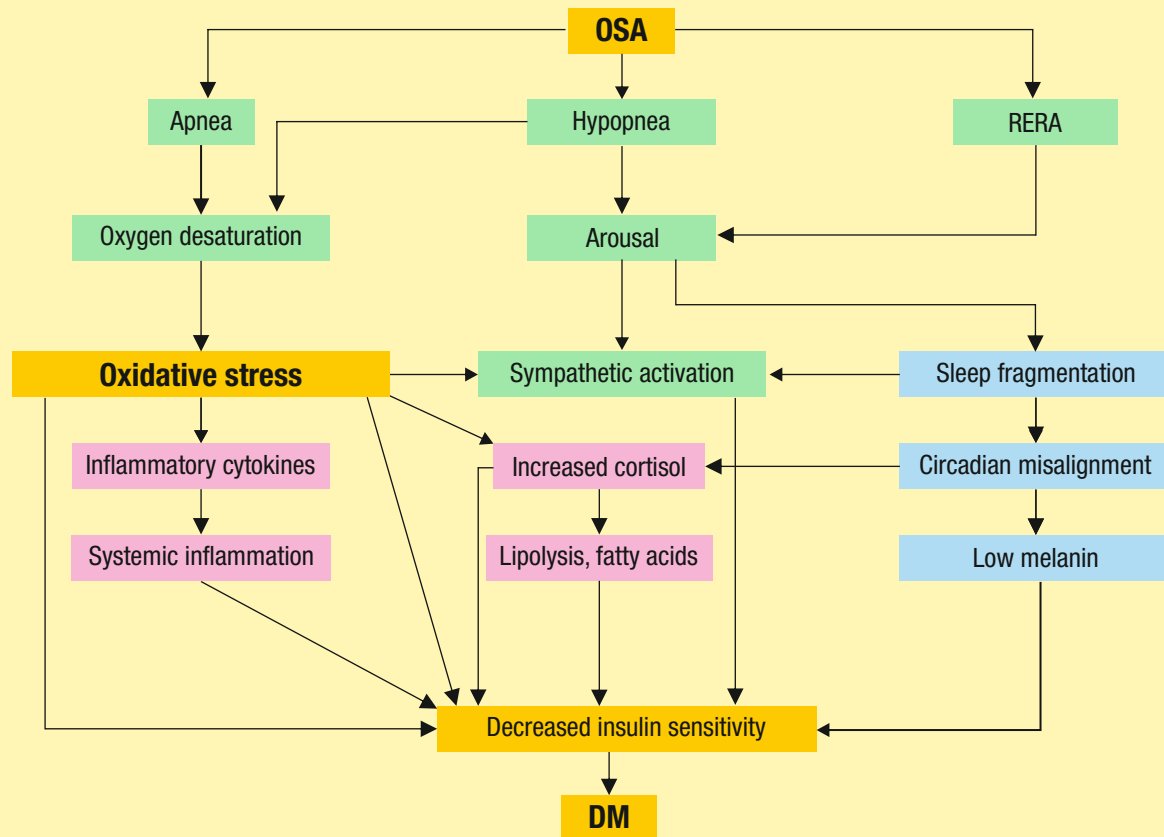
OSA occurs more commonly among individuals with obesity and diabetes and is associated with insulin resistance, polycythemia, hypercoagulability, metabolic dysfunction-associated steatotic liver disease (MASLD), and cardiovascular diseases (CVDs).

Obesity, OSA, sleep, and metabolic dysregulation are closely interlinked. Sleep disturbance may promote behavioral, metabolic, and/or hormonal changes with weight gain, which could increase diabetes risk. Glucose homeostasis is reported to be impaired by hypoxia that is associated with OSA. Individuals with chronic hypoxic respiratory disease have been shown to have significantly higher plasma glucose levels than healthy controls. Even intermittent hypoxia has been shown to reduce insulin sensitivity by 17% without increased insulin secretion. These findings suggest a link between OSA-induced hypoxia and a higher risk of type 2 diabetes.

Hypoxia may cause a stress-related increase in circulating cortisol concentrations as a result of hypothalamic-pituitary-adrenal axis activity. Cortisol interferes with glucose metabolism and increases the risk of diabetes through various mechanisms, such as inhibition of insulin secretion by modifying  $\beta$ -cell function, increasing hepatic gluconeogenesis, etc. Individuals with OSA also



exhibit hyperactivity of the sympathetic nervous system, characterized by elevated levels of epinephrine, norepinephrine, and urinary catecholamines following arousal. These catecholamines increase hepatic glucose production and reduce insulin-mediated glucose uptake and insulin sensitivity. Furthermore, amplified sympathetic activity has lipolytic effects, which increase non-esterified fatty acid levels.



**Abbreviations:** OSA: Obstructive sleep apnea; RERA: Respiratory effort-related arousal; DM: Diabetes mellitus

**Figure 4: Interaction of OSA and diabetes**

This worsens insulin sensitivity and glucose tolerance. An overview of different mechanisms associated with OSA and diabetes is presented in Figure 4. The prevalence of OSA in people with type 2 diabetes is higher than in the general population and increases further with OSA severity. On the other hand, uncontrolled glucose may desensitize the carotid body and pharyngeal dilator muscle, which promotes sleep-disordered breathing in OSA. Hence, OSA and diabetes have a bidirectional association. Lifestyle modification with weight loss is strongly recommended for both OSA and diabetes management.



### Resources:

1. Bloomgarden Z. Obstructive sleep apnea and diabetes. *J Diabetes*. 2023;15(11):916-919. doi:10.1111/1753-0407.13494
2. Song SO, He K, Narla RR, Kang HG, Ryu HU, Boyko EJ. Metabolic Consequences of Obstructive Sleep Apnea Especially Pertaining to Diabetes Mellitus and Insulin Sensitivity. *Diabetes Metab J*. 2019;43(2):144-155. doi:10.4093/dmj.2018.0256.
3. Agarwal L, Gupta A. Role of Orthodontist in Obstructive Sleep Apnea An Orthodontic Review. *J Orthod Endod*. 2016, 2:3. doi: 10.4172/2469-2980.100027

# The Impact of Respiratory Infections on Type 1 Diabetes Risk



## Dr. Swapnil Bhavsar

MBBS, FAIIDR (Diabetology), PGDM, PGADM,  
Certificate and Advance Certificate in  
Diabetes

Consultant Physician, Dr. Bhavsar's Diabetes  
Center, Surat

Type 1 diabetes (T1D) is a chronic autoimmune disease affecting more than nine million people globally. Among the environmental factors, viral infections are the most extensively investigated environmental factors associated with T1D and are believed to be the main triggers of islet autoimmunity (IA) and the progression to T1D,

especially during prenatal development and childhood.

While the majority of studies conducted on the infectious etiology of IA/T1D have focused primarily on viral infections of the pancreas and gut, respiratory tract infections (RTI), especially in the first year following birth, have also been studied as a possible risk factor for childhood T1D. At least 19 observational studies have looked at upper RTIs (rhinitis, pharyngitis, and laryngitis) and lower RTIs (pneumonia, bronchitis, and bronchiolitis) as possible triggers of IA/T1D development. RTIs and T1D were found to be significantly correlated in three retrospective case-control and cohort studies (Beyerlein A, *et al.* [2016], Lee HY, *et al.* [2015], and Ruiz PLD, *et al.* [2018]) but not in the other two (Ruiz PLD, *et al.* [2018] and Cardwell CR [2008]). However, these included infections with overt clinical symptoms only. Only one retrospective investigation (Ruiz PLD, *et al.* [2018]), which included molecular testing to confirm the infectious agent, reported a significant correlation between laboratory-confirmed influenza A (H1N1) and T1D, but not between clinically diagnosed H1N1 and T1D. Since none of these studies included IA testing, it was not possible to examine IA as a result of RTIs.



Some studies from Norway and Germany revealed a higher prevalence of IA in children with  $\geq 1$  RTI in the first four years and the first six months of life. These outcomes were supported by two large-scale birth cohort studies: (i) The Environmental Determinants of Diabetes in the Young (TEDDY), which found that the risk of IA increased by 5.6% for every RTI recorded in children up to 4 years of age and (ii) The Trial to Reduce Insulin-Dependent Diabetes Mellitus, which observed that upper RTIs in the first year of life was associated with IA. In contrast, other large investigations found no significant link between early-life RTIs and IA. These inconsistent results may be attributed in part to the limitations of assessing subjective data types, demanding

additional research using molecular approaches to confirm and describe diseases and the viruses that cause them.

While there is much epidemiological evidence supporting the significance of respiratory infections in T1D, there is a scarcity of data characterizing infectious agents at the molecular scale. This gap in the literature limits the identification of the specific infectious agents that link RTI and T1D. Hence, more in-depth research is needed to identify specific causative agents and develop appropriate subsequent management care.

## Resources:

1. Wu R, Mumtaz M, Maxwell AJ, *et al*. Respiratory infections and type 1 diabetes: Potential roles in pathogenesis. *Rev Med Virol*. 2023;33(2):e2429. doi:10.1002/rmv.2429
2. Lönnrot M, Lynch KF, Elding Larsson H, *et al*. Respiratory infections are temporally associated with initiation of type 1 diabetes autoimmunity: The TEDDY study [published correction appears in *Diabetologia*. 2018 Jan;61(1):254]. *Diabetologia*. 2017;60(10):1931-1940. doi:10.1007/s00125-017-4365-5
3. Beyerlein A, Donnachie E, Jergens S, Ziegler AG. Infections in early life and development of type 1 diabetes. *JAMA*. 2016; 315(17): 1899-1901.
4. Lee HY, Lu CL, Chen HF, Su HF, Li CY. Perinatal and childhood risk factors for early-onset type 1 diabetes: A population-based case-control study in Taiwan. *Eur J Publ Health*. 2015; 25(6): 1024-1029.
5. Ruiz PLD, Tapia G, Bakken IJ, *et al*. Pandemic influenza and subsequent risk of type 1 diabetes: A nationwide cohort study. *Diabetologia*. 2018; 61(9): 1996-2004.
6. Cardwell CR, Carson DJ, Patterson CC. No association between routinely recorded infections in early life and subsequent risk of childhood-onset Type 1 diabetes: A matched case-control study using the UK general practice research database. *Diabet Med*. 2008; 25(3): 261-267.





## Frequently Asked Questions on Diabetes and Respiratory Health



### Dr. Aditya Deshpande

MBBS, MD (Medicine), DM (Endocrinology)

Consultant Endocrinologist, Shantabai Deshpande Superspeciality Hospital, Baramati

1. My child was diagnosed with type 1 diabetes a few months ago and has recently developed a cough and cold due to the weather. I gave her some cough syrup, and ever since then, I have noticed her blood glucose levels spiking. Did I do something wrong? We are very worried. What should I do?

**Ans.** In type 1 diabetes, infections such as cough and cold can increase your blood sugar levels. Also, certain ingredients in the cough syrup can raise blood sugar levels. You must speak to your doctor if your sugar levels are running high with the prescribed medicines. Below are some measures you must follow, in addition to typical precautions like staying home if too sick.

- **Check medication ingredients:** Many cold and flu medications, such as cough syrups, contain sugar, which can raise blood sugar levels. Always read the ingredients label carefully. Consult your doctor or pharmacist for sugar-free or diabetes-safe over-the-counter medications. Keep a list of these recommended products for future reference.
- **Monitor blood sugar:** Test every 4 hours or as advised by your doctor. Adjust insulin as needed.
- **Test for ketones:** If blood glucose  $>250$  mg/dL, check for ketones and consult your doctor.
- **Stay hydrated:** Ensure regular fluid intake to prevent dehydration and aid recovery.
- **Monitor temperature:** Regularly check your child's temperature to track illness severity.

By following these guidelines, you can better manage your child's diabetes while dealing with a cold. Always stay in close communication with your doctor for personalized advice and support.

2. I am a 26-year-old with type 2 diabetes. Recently, I developed asthma. I used to have a cup of milk before bed and include curd in my meals. Now, due to my asthma, I have been told that I can't consume dairy. Is this true?



**Ans.** It's a common myth that individuals with asthma cannot consume dairy. While some people may believe dairy can increase mucus production and worsen asthma symptoms, scientific evidence does not support this for most individuals. You can continue to consume dairy products like milk and curd if they help you manage your blood sugar levels unless you have a specific dairy allergy or intolerance. Consult with your doctor and dietitian to create a balanced diet plan that suits both your diabetes and asthma management needs.

3. I am a 42-year-old male with diabetes for two years. I've developed a chronic cough and mild asthma due to pollution. Can diabetes worsen lung conditions? What precautions should I take?

**Ans.** Diabetes does weaken the immune system, heightening susceptibility to respiratory infections like pneumonia, etc., which can worsen existing lung conditions. Uncontrolled diabetes also impairs lung function and exacerbates respiratory symptoms.

To mitigate diabetes' impact on lung health, prioritize managing blood sugar levels through prescribed medications, regular monitoring, and lifestyle modifications like a balanced diet and exercise. Avoid smoking and reduce exposure to pollutants to minimize respiratory symptoms. Schedule regular medical check-ups, including pulmonary function tests, for early detection of respiratory issues. Get vaccinated against influenza and pneumonia to prevent infections. Manage comorbidities like obesity and hypertension through lifestyle changes and medication to improve overall lung function.



## Did You Know? Pranayama in Yoga Helps to Improve Lung Health

Yoga has gained increased popularity in recent times for promoting balance in individuals' physical, mental, emotional, and spiritual health. Hatha yoga, the most widely studied and practiced form of yoga in the modern world, focuses on two main aspects: "Asana" (physical postures) and "pranayama" (breathing exercises). "Pranayama" is a Sanskrit word formed by combining the terms "*prana*," which means life breath or vital energy, and "*ayama*," which refers to expansion/regulation or control. Pranayama, the yogic art of breathing, consists of purposeful changes to the breathing mechanism, usually performed while sitting down. These variations include rapid diaphragmatic breathing, slow, deep breathing, breathing via alternate nostrils, and breath holding and retention.



Several studies have thoroughly investigated the effect of pranayama on respiratory well-being. One such study that analyzed yogic breathing practices on lung functions of swimmers showed that pranayama enhances respiratory endurance in competitive swimmers. The study revealed that practicing pranayama for five days a week for half an hour, along with a daily physical exercise regimen for five days a week, enhanced autonomic reactivity and oxygen diffusion and reduced anxiety, resulting in lowered airway resistance, improved respiratory muscle endurance, increased strokes per breath among competitive swimmers.

The research team from another study concluded that pranayama is a valuable adjunct treatment and can serve as an effective rehabilitation program for individuals with chronic obstructive pulmonary disease (COPD). Results demonstrated that after three months of practice, pranayama can enhance subjective health, reduce disease severity, and improve functional status in COPD patients.

Six studies examined the effects of pranayama on patients with bronchial asthma, along with additional individual studies focusing on patients with COPD and pleural effusion. Bhatt and Rampallivar (2016) studied the impact of pranayama on pulmonary functions in 80 patients with bronchial asthma. The patients were divided equally into two groups, with the intervention group practicing pranayama in addition to their medication for three months. Measurements of pulse rate (PR), systolic blood pressure (SBP), diastolic blood pressure (DBP), respiratory rate, peak expiratory flow rate (PEFR), forced expiratory volume in 1 second (FEV<sub>1</sub>), and forced vital capacity (FVC) were taken for all participants. The intervention group showed significant improvements in PR, SBP, FVC, PEFR, and FEV<sub>1</sub> in post-test values compared to pre-test values, with no significant changes observed in the control group.

Another study revealed that after two months of consistent yoga practice, participants showed enhanced pulmonary functions among healthy individuals and a potential preventive effect against respiratory ailments. The findings suggest that these positive impacts of pranayama could serve as supplementary therapy for various respiratory conditions.



### Resources:

1. Jayawardena R, Ranasinghe P, Ranawaka H, Gamage N, Dissanayake D, Misra A. Exploring the Therapeutic Benefits of Pranayama (Yogic Breathing): A Systematic Review. *Int J Yoga*. 2020;13(2):99-110. doi:10.4103/ijoy.IJOY\_37\_19 <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7336946/>
2. Hakked CS, Balakrishnan R, Krishnamurthy MN. Yogic breathing practices improve lung functions of competitive young swimmers. *J Ayurveda Integr Med*. 2017;8(2):99-104. doi:10.1016/j.jaim.2016.12.005 <https://www.sciencedirect.com/science/article/pii/S0975947616300675>
3. Karthik PS, Chandrasekhar M, Ambareesha K, Nikhil C. Effect of pranayama and suryanamaskar on pulmonary functions in medical students. *J Clin Diagn Res*. 2014;8(12):BC04-BC6. doi:10.7860/JCDR/2014/10281.5344 <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4316242/#:~:text=The%20daily%20practice%20could%20also,and%20forced%20expiratory%20lung%20volumes-study link>
4. Gupta A, Gupta R, Sood S, Arkham M. Pranayam for Treatment of Chronic Obstructive Pulmonary Disease: Results From a Randomized, Controlled Trial. *Integr Med (Encinitas)*. 2014;13(1):26-31. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4684118/>
5. Bhatt A, Rampallivar S. Effect of pranayam on ventilatory functions in patients of bronchial asthma. *J Evol Med Dent Sci*. 2016;5(28):1453-1456.



# Dia-Games

## Word search

Words can be found in any direction (including diagonals) and can overlap each other. Use the word bank below.

L	A	J	E	Q	T	B	I	H	Z	J	B	A	P	N	E	A	C	E	G
T	U	B	O	V	E	E	O	Q	A	T	A	K	J	S	V	I	G	C	C
A	Z	Q	P	U	D	T	M	G	M	K	I	U	S	N	Y	Y	G	T	S
L	N	I	B	B	A	Y	P	L	L	O	M	M	Z	W	N	C	M	W	V
A	O	M	Y	S	V	H	K	U	S	O	E	X	S	K	F	E	N	K	Z
Z	F	C	S	D	O	U	B	C	I	I	C	P	T	B	J	H	A	N	G
X	V	U	B	J	D	J	F	O	T	L	Y	I	L	O	E	V	L	A	H
P	U	E	Q	F	T	M	Y	C	I	W	L	E	F	F	U	S	I	O	N
V	X	P	X	L	A	V	Z	O	L	M	G	B	N	S	X	L	F	O	U
H	T	A	M	H	T	S	A	R	O	G	R	Q	S	N	D	A	C	R	X
O	P	J	J	Y	Q	E	A	T	I	J	E	P	L	P	V	R	H	B	P
F	H	X	Y	B	D	S	P	I	H	R	P	R	E	Y	A	U	K	S	W
M	O	C	Q	P	Y	K	P	C	C	L	Y	A	E	Q	V	E	M	P	D
L	J	X	C	R	K	M	T	O	N	P	H	N	P	D	Y	L	U	Q	T
B	F	M	D	K	P	B	O	I	O	N	J	A	Q	A	M	P	V	R	I
Y	S	L	G	J	E	I	H	D	R	W	A	Y	A	W	E	W	M	D	I
T	H	L	L	G	T	Q	N	S	B	F	V	A	E	B	N	Q	S	K	P
J	V	U	P	U	L	M	O	N	A	R	Y	M	P	A	L	B	L	H	S
W	X	T	R	Q	S	Y	G	P	F	X	Q	A	F	S	S	X	X	V	T
S	I	S	O	L	U	C	R	E	B	U	T	L	P	F	E	P	K	N	B

1. Asthma
2. Effusion
3. Sleep
4. Pranayama
5. Pulmonary
6. Bronchiolitis
7. Apnea
8. Pleural
9. Tuberculosis
10. Alveoli
11. Glucocorticoids
12. Hyperglycemia

L	A	J	E	Q	T	B	I	H	Z	J	B	A	P	N	E	A	C	E	G
T	U	B	O	V	E	E	O	Q	A	T	A	K	J	S	V	I	G	C	C
A	Z	Q	P	U	D	T	M	G	M	K	I	U	S	N	Y	Y	G	T	S
L	N	I	B	B	A	Y	P	L	L	O	M	M	Z	W	N	C	M	W	V
A	O	M	Y	S	V	H	K	U	S	O	E	X	S	K	F	E	N	K	Z
Z	F	C	S	D	O	U	B	C	I	I	C	P	T	B	J	H	A	N	G
X	V	U	B	J	D	J	F	O	T	L	Y	I	L	O	E	V	L	A	H
P	U	E	Q	F	T	M	Y	C	I	W	L	E	F	F	U	S	I	O	N
V	X	P	X	L	A	V	Z	O	L	M	G	B	N	S	X	L	F	O	U
H	T	A	M	H	T	S	A	R	O	G	R	Q	S	N	D	A	C	R	X
O	P	J	J	Y	Q	E	A	T	I	J	E	P	L	P	V	R	H	B	P
F	H	X	Y	B	D	S	P	I	H	R	P	R	E	Y	A	U	K	S	W
M	O	C	Q	P	Y	K	P	C	C	L	Y	A	E	Q	V	E	M	P	D
L	J	X	C	R	K	M	T	O	N	P	H	N	P	D	Y	L	U	Q	T
B	F	M	D	K	P	B	O	I	O	N	J	A	Q	A	M	P	V	R	I
Y	S	L	G	J	E	I	H	D	R	W	A	Y	A	W	E	W	M	D	I
T	H	L	L	G	T	Q	N	S	B	F	V	A	E	B	N	Q	S	K	P
J	V	U	P	U	L	M	O	N	A	R	Y	M	P	A	L	B	L	H	S
W	X	T	R	Q	S	Y	G	P	F	X	Q	A	F	S	S	X	X	V	T
S	I	S	O	L	U	C	R	E	B	U	T	L	P	F	E	P	K	N	B

## NOTES

Choose **GREEN**

# Glycomet®-GP

Metformin Hydrochloride 500/850/1000 mg SR + Glimepiride 0.5/1/2/3/4 mg

Widely available range for Individualised needs of the patients  
Across T2DM Continuum



**Glycomet®-GP 1**  
Metformin Hydrochloride 500 mg SR + Glimepiride 1 mg

**Glycomet®-GP 3/850**  
Metformin Hydrochloride 850 mg SR + Glimepiride 3 mg

**Glycomet®-GP 0.5**  
Metformin Hydrochloride 500 mg SR + Glimepiride 0.5 mg

**Glycomet®-GP 2/850**  
Metformin Hydrochloride 850 mg SR + Glimepiride 2 mg

**Glycomet®-GP 2**  
Metformin Hydrochloride 500 mg SR + Glimepiride 2 mg

**Glycomet®-GP 0.5 FORTE**  
Metformin Hydrochloride 1000 mg SR + Glimepiride 0.5 mg

**Glycomet®-GP 4 FORTE**  
Metformin Hydrochloride 1000 mg SR + Glimepiride 4 mg

**Glycomet®-GP 1 FORTE**  
Metformin Hydrochloride 1000 mg SR + Glimepiride 1 mg

**Glycomet®-GP 3 FORTE**  
Metformin Hydrochloride 1000 mg SR + Glimepiride 3 mg

**Glycomet®-GP 2 FORTE**  
Metformin Hydrochloride 1000 mg SR + Glimepiride 2 mg

## Abridged Prescribing Information

**Active Ingredients:** Metformin hydrochloride (as sustained release) and glimepiride tablets. **Indication:** For the management of patients with type 2 diabetes mellitus when diet, exercise and single agent (glimepiride or metformin alone) do not result in adequate glycaemic control. **Dosage and Administration:** The recommended dose is one tablet daily during breakfast or the first main meal. Each tablet contains a fixed dose of glimepiride and Metformin Hydrochloride. The highest recommended dose per day should be 8 mg of glimepiride and 2000mg of metformin. Due to prolonged release formulation, the tablet must be swallowed whole and not crushed or chewed. **Adverse Reactions:** For Glimepiride: hypoglycaemia may occur, which may sometimes be prolonged. Occasionally, gastrointestinal (GI) symptoms such as nausea, vomiting, sensations of pressure or fullness in the epigastrium, abdominal pain and diarrhea may occur. Hepatitis, elevation of liver enzymes, cholestasis and jaundice may occur; allergic reactions or pseudo allergic reactions may occur occasionally. For Metformin: GI symptoms such as nausea, vomiting, diarrhea, abdominal pain, and loss of appetite are common during initiation of therapy and may resolve spontaneously in most cases. Metallic taste, mild erythema, decrease in Vit B12 absorption, very rarely lactic acidosis, Hemolytic anemia. Reduction of thyrotropin level in patients with hypothyroidism. Hypomagnesaemia in the context of diarrhea, Encephalopathy, Photosensitivity, hepatobiliary disorders. **Warnings and Precautions:** For Glimepiride: Patient should be advised to report promptly exceptional stress situations (e.g., trauma, surgery, febrile infections), blood glucose regulation may deteriorate, and a temporary change to insulin may be necessary to maintain good metabolic control. Metformin Hydrochloride may lead to Lactic acidosis; in such cases metformin should be temporarily discontinued and contact with a healthcare professional is recommended. Sulfonylureas have an increased risk of hypoglycaemia. Long-term treatment with metformin may lead to peripheral neuropathy because of decrease in vitamin B12 serum levels. Monitoring of the vitamin B12 level is recommended. Overweight patients should continue their energy-restricted diet, usual laboratory tests for diabetes monitoring should be performed regularly. **Contraindications:** Hypersensitivity to the active substance of glimepiride & Metformin or to any of the excipients listed. Any type of acute metabolic acidosis (such as lactic acidosis, diabetic ketoacidosis, diabetic pre-coma). Severe renal failure (GFR <30 ml/min). In pregnant women. In lactating women. Acute conditions with the potential to alter renal function (dehydration, severe infection, shock, intravascular administration of iodinated contrast agents); acute or chronic disease which may cause tissue hypoxia (cardiac or respiratory failure, recent myocardial infarction, shock); hepatic insufficiency; acute alcohol intoxication; alcoholism. **Use in a special population:** Pregnant Women: Due to a lack of human data, drugs should not be used during pregnancy. Lactating Women: It should not be used during breastfeeding. Pediatric Patients: The safety and efficacy of drugs has not yet been established. Renal impairment: A GFR should be assessed before initiation of treatment with metformin containing products and at least annually thereafter. In patients at increased risk of further progression of renal impairment and in the elderly, renal function should be assessed more frequently, e.g. every 3-6 months.

**Additional information is available on request.**

**Last updated:** March 13, 2023

\*In case of any adverse events, kindly contact: [pv@usv.in](mailto:pv@usv.in)

For the use of registered medical practitioner, hospital or laboratory.\*



**USV Private Limited**



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*Win Life*

For screening people with High & Moderate Risk of Diabetes

## Indian Diabetes Risk Score



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TO KNOW  
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In T2DM Across Continuum,

Choose

# Glycomet®-GP

Metformin Hydrochloride 500/850/1000 mg SR + Glimepiride 0.5/1/2/3/4 mg

**G**reen energy

**R**ecycle, Reduce  
& Reuse

**E**r Neutralality

**E**co-friendly  
manufacturing Facility

**N**o Human Intervention

**T**echnology innovation

**E**nergy efficient

**C**arbon footprint  
reduction

**H**igh Standard Quality

ICH: International Council of  
Harmonisation



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**GREENTECH**

\* AIOCD FEB'24 MAT data # Data on File

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**Corvette Team**

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