Interpretation of Key Updates to the 2024 GOLD Global Strategy for the Diagnosis, Treatment, Management, and Prevention of Chronic Obstructive Pulmonary Disease (Post-Print)

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Abstract

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2024 revision was released on 2023-11-13. Overall, its definition, diagnosis, assessment, and treatment of chronic obstructive pulmonary disease (hereinafter referred to as COPD) remain the same as GOLD 2023, but revisions/expansions have been made in 10 aspects, including expanding the concept of Preserved Ratio Impaired Spirometry (PRISm), adding a section on lung hyperinflation, adding instructions on conducting spirometry before inhaled bronchodilator use, adding a section on screening for target populations with COPD, updating the description of blood eosinophil count, updating the section on interstitial lung abnormalities, revising the smoking cessation section, updating recommended vaccines for COPD patients, expanding the section on management of inhaled therapy, and adding pharmacotherapy for smoking cessation. This article provides a brief introduction and interpretation of the updated content.

Full Text

Interpretation of Key Updates in the Global Strategy for the Diagnosis, Treatment, Management, and Prevention of Chronic Obstructive Pulmonary Disease (GOLD) 2024

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Abstract The Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2024 report was released on November 13, 2023. Overall, the definition, diagnosis, assessment, and therapy of chronic obstructive pulmonary disease (COPD) remain consistent with GOLD 2023, but the document includes revisions and expansions across ten aspects: expanded concepts on preserved ratio impaired spirometry (PRISm), a new section on lung hyperinflation, clarification on pre-bronchodilator spirometry, a new section on screening for COPD in targeted populations, updated guidance on blood eosinophil counts, updated content on interstitial lung abnormalities, revised smoking cessation sections, updated vaccination recommendations aligned with current CDC guidelines, expanded management of inhaled therapy, and new pharmacotherapies for smoking cessation. This article introduces and interprets these updated contents.

[Key words] Pulmonary disease, chronic obstructive; Hyperinflation; Pulmonary function; Screening for COPD; Smoking cessation; Guidebooks

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The GOLD 2024 report represents a revision of GOLD 2023, incorporating 148 new references published between January 2022 and July 2023, including ten studies from Chinese researchers. Released ahead of the 22nd World COPD Day (November 15, 2023) under the theme "Breathing is Life - Act Earlier," the report maintains its six-chapter structure but merges the former Chapter 3 (Evidence Supporting Prevention and Maintenance Therapy) with Chapter 4 (Management of Stable COPD) into a consolidated Chapter 3 (Prevention and Management of COPD) to eliminate redundancy. The most significant content revisions and additions span ten key areas: (1) expanded concepts on preserved ratio impaired spirometry (PRISm); (2) addition of lung hyperinflation content; (3) clarification on conducting spirometry before bronchodilator administration; (4) addition of targeted COPD screening sections; (5) updated guidance on blood eosinophil counts in initial assessment; (6) updated interstitial lung abnormalities content; (7) revised smoking cessation sections; (8) updated vaccination recommendations aligned with CDC guidelines; (9) expanded management of inhaled therapy; and (10) new pharmacotherapies for smoking cessation [1]. This article outlines the key points and updates in GOLD 2024.

1.1.1 Key Points

COPD is a heterogeneous disease characterized by chronic respiratory symptoms (dyspnea, cough, sputum production, exacerbations) due to airway (bronchitis, bronchiolitis) and/or alveolar abnormalities (emphysema), resulting in persistent, progressive airflow limitation. Risk factors follow a GETomics framework (Genes-Environment-Events across the life course), causing lung tissue destruction and altering normal lung development or aging processes. The primary environmental exposure is tobacco smoke and inhalation of toxic particles and gases from indoor/outdoor air pollution, though other environmental and host factors (including abnormal lung development and accelerated aging) can con-

tribute. The most relevant (though rare) genetic factor is SERPINA1 mutation causing alpha-1 antitrypsin deficiency (AATD); other genetic variants associate with lung function decline and COPD risk but with smaller effect sizes. COPD diagnosis requires post-bronchodilator spirometry showing FEV1/FVC < 0.7, indicating incompletely reversible airflow limitation. Some patients may have respiratory symptoms and/or structural lung destruction (emphysema) and/or physiological abnormalities (e.g., reduced FEV1, gas trapping, hyperinflation, impaired diffusing capacity) and/or rapid FEV1 decline without airflow limitation (post-bronchodilator FEV1/FVC ≥ 0.7). These individuals are classified as pre-COPD; PRISm identifies those with preserved ratio but impaired spirometry. Both groups are at high risk for developing airflow limitation, though not all will progress. Typical COPD symptoms include dyspnea, activity limitation, and/or cough with or without sputum, with acute worsening termed COPD exacerbations requiring preventive and therapeutic strategies. COPD patients frequently have comorbidities affecting disease status and prognosis that require targeted treatment. Comorbidities can mimic and promote exacerbations. COPD is a common preventable and treatable disease with serious underdiagnosis and misdiagnosis, making early correct diagnosis a major public health priority. Early-life exposures, including tobacco smoke and other environmental factors, influence COPD development in young adults, and attention to pre-COPD and PRISm facilitates disease prevention, early diagnosis, and prompt appropriate intervention.

1.1.2 Update Points

GOLD 2024 revised Figure 1, updating FEV1 trajectory changes across the life course while expanding PRISm concepts and adding content on lung hyperinflation.

1.2 Lung Function Trajectories: Development and Aging

The lungs are not fully developed at birth. Lung growth and maturation continue until ages 20-25 (earlier in females), when lung function reaches its peak [Figure 1: see original paper] [4]. This is followed by a relatively short, poorly defined plateau phase, then a mild decline due to physiological lung aging. This normal trajectory can be altered by processes during pregnancy, birth, childhood, and adolescence that affect lung growth (and thus peak lung function) and/or shorten the plateau phase and/or accelerate the aging phase (increasing the normal rate of age-related lung function decline). Reduced peak lung function measured by spirometry can identify individuals at increased COPD risk. A large study and meta-analysis confirmed positive associations between birth weight and adult FEV1 [5]. Early-life "childhood disadvantage factors" are key determinants of adult lung function changes. One study evaluating three independent longitudinal cohorts (Framingham, Copenhagen, and Lovelace) found that approximately 50% of patients developed COPD through accelerated FEV1 decline over time (the traditional Fletcher and Peto model) [6], while the other

50% developed COPD due to abnormal lung growth and development (normal decline from a lower peak; [Figure 1: see original paper]) [7].

Age is commonly listed as a COPD risk factor, with physiological lung function decline occurring with aging. However, it remains unclear whether normal aging causes COPD or whether age reflects cumulative lifetime exposure. Airway and parenchymal aging exhibits structural changes associated with COPD, with evidence that COPD accelerates aging [8]. A prospective study following COPD patients for over 10 years showed correlations between accelerated telomere shortening (a marker of aging) and progressive deterioration in gas exchange, lung hyperinflation, and extrapulmonary involvement [9]. Additionally, sustained telomere shortening increased all-cause mortality risk during the observation period. Age-related DNA epigenetic changes in immune cells also correlate with increased exacerbation and mortality risk in COPD patients [10]. In the Tasmanian Longitudinal Health Study, mixed (low FVC and low FEV1/FVC) and pure obstructive (low FEV1/FVC) lung function patterns showed highest COPD prevalence at age 53 (37% and 22%, respectively) [11].

The term "dysanapsis" refers to a mismatch between airway tree caliber and lung volume relative to body size, first described by TODISCO et al. [12] approximately 50 years ago based on variability in maximal expiratory flow in healthy adults. While understanding of dysanapsis origins and clinical significance remains limited, recent CT studies show: (1) the condition is common in general populations [13]; (2) it correlates with FEV1/FVC in early adulthood [14]; (3) in healthy adult donor lungs, central airway dysanapsis (detectable by CT) extends to peripheral airways (not visible on CT) [14]; (4) dysanapsis associates with baseline airflow obstruction and COPD risk independent of age, sex, height, and race-ethnicity, but not with lung function decline over time [15], consistent with trajectories showing low peak lung function in early adulthood followed by normal decline (accounting for 50% of COPD in older adults) [7]; (5) dysanapsis may contribute to obstructive lung disease pathophysiology and aerosol drug deposition [15]; and (6) the mechanisms remain unclear. It is uncertain whether this results from genetic predisposition, in-utero exposure to harmful particles or pathogens, prematurity, low birth weight, neonatal lung injury, childhood recurrent respiratory infections, or multiple concurrent factors. Factors affecting bronchial tree growth in early life and those affecting homeostasis in later life appear associated with dysanapsis. Notably, radiation-free (or low-dose) methods are needed to quantify pediatric lung structure when studying childhood dysanapsis etiology. Thus, two major biological mechanisms causing adult COPD include abnormal lung development and accelerated age-related lung function decline, which can coexist. Some individuals have below-normal lung function in childhood that worsens during adolescence; some adults have above-normal early lung function that declines with smoking and other factors. Nevertheless, these individuals may later show "pseudo-normal"spirometry (symptoms and structural destruction like emphysema present but normal lung function).

1.3 Expanded PRISm Concepts

PRISm describes individuals with preserved ratio (post-bronchodilator FEV1/FVC ≥ 0.7) but impaired spirometry (post-bronchodilator FEV1 $< 80\%$ predicted). Population-based studies report PRISm prevalence of 7.1%-11.0% [16], while in current and former smokers (e.g., COPDGene cohort), prevalence is 10.4%-11.3% [17]. PRISm prevalence is particularly high in current and former smokers and associates with high and low BMI, female sex, obesity, and multimorbidity. PRISm correlates with increased risks of cardiopulmonary disease, all-cause and cardiovascular mortality, hospitalization, and airway obstruction.

PRISm is not always a stable phenotype. Over time, 20%-30% of PRISm patients transition to normal spirometry or airflow obstruction. The most important predictors of PRISm progression to COPD are lower baseline FEV1% and FEV1/FVC, older age, current smoking, female sex, and longer forced exhalation time on second assessment. Despite growing literature, substantial knowledge gaps remain regarding PRISm pathogenesis and treatment. Additionally, not all pre-COPD or PRISm individuals develop fixed airflow obstruction over time, but all should be considered "patients" (as they already have symptoms and/or functional and/or structural abnormalities) and deserve care and treatment. The challenge is that no evidence currently indicates the best treatment approach for these patients.

1.4 Lung Hyperinflation

Lung hyperinflation occurs when lung gas volume at end-expiration exceeds normal values. It correlates with clinical manifestations in COPD patients, causing dyspnea, impaired exercise capacity, increased hospitalization risk, respiratory failure, and mortality. In COPD, hyperinflation results from loss of lung elasticity and expiratory flow limitation. Expiratory flow limitation occurs when expiratory flow generated during spontaneous breathing reaches the maximal flow possible at that lung volume, caused by combined parenchymal destruction (emphysema) and airway abnormalities (mucus plugging, airway edema, increased bronchial tone, airway wall remodeling). Hyperinflation occurs at rest (static hyperinflation from loss of elastic recoil in emphysema) and/or during exercise (dynamic hyperinflation from flow limitation) when ventilatory demands increase and expiratory time decreases.

Hyperinflation is common in COPD, even in patients with mild obstruction at rest, and more prevalent during exercise. In moderate-to-severe obstruction, dynamic hyperinflation correlates more strongly with impaired diffusing capacity, small airway obstruction severity, and higher ventilatory response to exercise than FEV1. Lung volumes assessed by plethysmography or gas dilution methods (helium dilution or nitrogen washout) are reference indicators for hyperinflation presence and severity, though values may differ due to measurement of compressible versus communicating gas volumes. Inspiratory capacity measurements at

rest and during exercise are indirect indicators of end-expiratory lung volume increase, showing static and/or dynamic hyperinflation. Chest imaging can also detect hyperinflation but lacks standardization.

Hyperinflation can be treated with bronchodilators, oxygen therapy, heliox, pulmonary rehabilitation, pursed-lip breathing, inspiratory muscle training, or lung volume reduction surgery/bronchoscopic lung volume reduction for severe emphysema.

2.1.1 Key Points

COPD should be considered in patients with dyspnea, chronic cough or sputum, recurrent lower respiratory infections, and/or exposure risk factors. Spirometry is essential for diagnosis; FEV1/FVC < 0.70 confirms persistent airflow limitation. COPD assessment aims to determine airflow limitation severity, disease impact on health status, and future adverse risk (exacerbations, hospitalization, or death) to guide therapy. COPD patients with persistent symptoms despite initial treatment should undergo further assessment including lung volumes, diffusing capacity, exercise testing, and/or chest imaging. COPD patients commonly have comorbidities including cardiovascular disease, skeletal muscle dysfunction, metabolic syndrome, osteoporosis, depression, anxiety, and lung cancer, which affect hospitalization and mortality regardless of airflow limitation severity. All COPD patients should be actively screened for comorbidities and receive appropriate treatment.

2.1.2 Update Points

GOLD 2024 emphasizes pre-bronchodilator spirometry, targeted COPD screening, updated blood eosinophil count guidance, and interstitial lung abnormalities in imaging sections. The COPD diagnosis and assessment flowchart in GOLD 2024 is shown in [Figure 2: see original paper].

2.2 Pre-bronchodilator Spirometry

Previous GOLD guidelines recommended post-bronchodilator spirometry for COPD diagnosis, considering these measures more suitable for diagnosing airflow limitation with high reproducibility and for excluding asthma. However, it is now recognized that bronchodilator responsiveness has little diagnostic value in distinguishing asthma from COPD, while pre-bronchodilator measures show higher repeatability and airway obstruction after bronchodilator is uncommon. Post-bronchodilator testing is more time-consuming and may discourage clinicians from ordering spirometry. The latest GOLD guideline states that pre-bronchodilator spirometry can be used for initial assessment of airflow limitation in symptomatic patients. If pre-bronchodilator results show no obstruction, post-bronchodilator testing is unnecessary unless COPD is strongly suspected clinically. In such cases, FEV1/FVC may be < 0.7, requiring further investigation and follow-up including repeat spirometry. If pre-bronchodilator results

already show airflow limitation, post-bronchodilator values should be used for COPD diagnosis. Individuals with pre-bronchodilator FEV1/FVC < 0.7 but post-bronchodilator FEV1/FVC ≥ 0.7 have high risk for future COPD progression and require close follow-up.

2.3 Targeted COPD Screening

The U.S. Preventive Services Task Force (USPSTF) recommends against screening asymptomatic individuals based on systematic reviews of clinical trials in patients with mild or no symptoms [18]. However, this does not apply to high-risk populations such as those requiring annual low-dose chest CT (LDCT) for lung cancer detection or those with imaging abnormalities (emphysema, airway wall thickening, bronchiectasis) and respiratory symptoms.

2.3.1 Screening COPD Using Lung Cancer Imaging USPSTF recommends annual LDCT for individuals aged 50-80 with $ $20 pack-year smoking history for early lung cancer diagnosis. Clinical trials show annual LDCT significantly improves survival [19]. Lung cancer and COPD share risk factors, and COPD is an independent lung cancer risk factor and key comorbidity affecting survival. Therefore, evaluating symptoms and performing spirometry in individuals undergoing LDCT screening presents a unique opportunity to simultaneously screen for COPD.

Studies assessing COPD symptoms and spirometry during lung cancer screening report airflow obstruction prevalence of 34%-57%, emphysema prevalence of 68%-73%, and newly diagnosed COPD prevalence of 67%. Male sex, younger age, shorter smoking duration, and being asymptomatic correlate with previously undiagnosed airflow obstruction [20]. Previously undiagnosed COPD patients have mild symptoms, but symptom prevalence remains high, with over 50% experiencing symptoms. Some studies report COPD underdiagnosis rates up to 90% in lung cancer screening populations [21].

In lung cancer screening cohorts, over half of patients with visually evident emphysema have airflow obstruction [22]. Quantitative densitometry can also detect COPD, with sensitivity and specificity depending on selected thresholds. In the National Lung Screening Trial (NLST), a 1% quantitative emphysema threshold in individuals >65 years showed sensitivity of 65% in women and 75% in men, with specificity exceeding 70% and 65%, respectively [23].

2.3.2 Screening COPD Using Incidental Chest Imaging Abnormalities Factors beyond smoking increase COPD risk (e.g., development, genetics, environmental exposures, childhood infections). Such patients may undergo chest imaging for respiratory symptoms. These populations are often younger with minimal or no smoking history. Unlike annual LDCT lung cancer screening populations, CT scans themselves can identify individuals at increased COPD risk in non-lung-cancer screening contexts, prompting consideration of spirometry. Emphysema, a hallmark of COPD, is easily detected on chest imaging through

radiologist visual inspection or quantitative lung density. Imaging can also identify other COPD abnormalities including gas trapping, airway wall thickening, and mucus plugging. These abnormalities not only suggest possible airflow obstruction but also predict accelerated lung function decline and worse quality of life. While quantitative LDCT analysis is often impractical clinically, the presence of emphysema and other airway abnormalities should raise clinical suspicion for COPD, prompting detailed symptom assessment and consideration of spirometry.

GOLD 2024 recommends further spirometry evaluation for targeted patients undergoing lung cancer screening and those with incidental airway imaging abnormalities, as these patients currently miss opportunities for spirometry testing.

2.3.3 Updated Blood Eosinophil Count Guidance Blood eosinophil counts can predict ICS efficacy for preventing future exacerbations, so GOLD recommends using them in ICS management. Higher blood eosinophil counts in COPD patients correlate with lung eosinophil numbers and Type 2 inflammation marker expression [24]. Differences in airway inflammation may explain varying ICS responses at different eosinophil counts. In large primary care populations, blood eosinophil counts show good reproducibility. While greater variability is observed at higher thresholds, better repeatability is seen at lower thresholds (e.g., 100 cells/ L) [25]. Blood eosinophil counts help clinicians assess the likelihood of beneficial preventive responses when adding ICS to regular bronchodilator therapy and can be used as a biomarker alongside clinical assessment for ICS decisions.

2.3.4 Interstitial Lung Abnormalities Parenchymal fibrosis or inflammation is common on chest CT in both smokers and non-smokers. When found incidentally in patients without interstitial lung disease (ILD), these are termed interstitial lung abnormalities (ILA). In older adults (>60 years), ILA prevalence is 4%-9%, encompassing a spectrum from subclinical to clinical disease [26]. Among 4,360 COPDGene participants, 8% had ILA, half meeting suspected ILD criteria (evident fibrosis on CT, FVC <80% predicted, or diffusing capacity <70% predicted) [27-28]. Suspected ILD associates with increased respiratory symptoms and mortality [28]. Fibrotic ILA (traction bronchiectasis, architectural distortion, honeycombing) is more likely to progress and associate with poor outcomes, especially when combined with emphysema. Given these clinical correlations, multiple studies support clinical evaluation, risk stratification, and follow-up monitoring for symptomatic individuals.

3.1.1 Key Points

Smoking cessation is paramount. Pharmacotherapy and nicotine replacement therapy improve long-term quit rates. Legislative smoking bans and counseling by healthcare professionals increase cessation rates. The efficacy and safety of e-cigarettes as cessation aids remain uncertain. The primary COPD treatment

goals are symptom reduction and decreasing future exacerbation risk. Stable COPD management should be based primarily on clinical symptoms and exacerbation history. Pharmacotherapy alleviates symptoms, reduces exacerbation frequency and severity, improves health status and exercise tolerance, slows lung function decline, and reduces mortality. Each treatment regimen should be individualized based on symptom severity, exacerbation risk, adverse effects, comorbidities, drug availability and cost, and patient response, preference, and ability to use delivery devices. Inhaler technique requires regular assessment. COVID-19 vaccination effectively prevents infection; COPD patients should receive it per national recommendations. Influenza and pneumococcal vaccines reduce lower respiratory infection rates. CDC recommends Tdap (diphosphorylated pertussis/tetanus/diphtheria) vaccination for adolescent-unvaccinated COPD patients and herpes zoster vaccine for those aged $ $50. Pulmonary rehabilitation combining exercise training with health education improves exercise capacity, symptoms, and quality of life across COPD severity levels. Long-term oxygen therapy improves survival in severe chronic hypoxemia [PaO2 $ $55 mmHg or <60 mmHg with cor pulmonale or secondary polycythemia]. Routine long-term oxygen therapy is not recommended for stable COPD patients with resting or exercise-induced moderate desaturation, though individual factors must be considered. Non-invasive ventilation reduces mortality and prevents rehospitalization in patients with severe chronic hypercapnia and acute respiratory failure history. Surgical or bronchoscopic interventions may benefit selected patients with advanced emphysema unresponsive to medical therapy. Palliative care effectively manages advanced COPD symptoms.

3.1.2 Update Points

GOLD 2024 merged former Chapters 3 and 4 into "Prevention and Management of COPD," with revisions to smoking cessation, updates on indoor/outdoor air pollution, occupational exposure, RSV vaccine recommendations, inhaled therapy management, and smoking cessation pharmacotherapy. The principles of pharmacological treatment during initial and follow-up COPD management are shown in [Figure 3: see original paper] and [Figure 4: see original paper].

3.2 Revised Smoking Cessation Section

Smoking cessation is the most effective treatment for COPD patients who continue smoking. Healthcare professionals play a key role in cessation counseling and intervention, using every opportunity to encourage quitting. Cessation has the greatest impact on COPD natural history and improves daily symptoms while reducing exacerbation frequency. Quitting is more challenging for COPD smokers due to higher nicotine dependence, lower self-efficacy, and less confidence, with depression being more common and contributing to failed quit attempts. Despite these challenges, long-term quit rates of 14%-27% are achievable with effective resource allocation [29]. Cessation treatment should be tailored to individual needs and tobacco dependence level. Combined counseling

and pharmacotherapy is most effective. Nicotine dependence should be assessed in all patients; high dependence indicators include desire to smoke within 30 minutes of waking, nighttime smoking, $ $20 cigarettes daily, Fagerström score of 7-10, or Smoking Dependence Index score of 5-6 [30]. Legislative smoking bans effectively increase quit rates and reduce secondhand smoke exposure.

3.3 Indoor and Outdoor Air Pollution

Reducing household and outdoor air pollution exposure requires integrated public policy, local and national resources, cultural change, and individual protective measures. Reducing biomass smoke exposure is a key target for lowering global COPD prevalence. Efficient ventilation, non-polluting stoves, and similar interventions should be recommended.

3.4 Occupational Exposure

No studies have proven whether interventions reducing occupational exposure also lessen COPD burden, but patients should avoid continued exposure to potential irritants (dust, smoke, gases) when possible.

3.5 Respiratory Syncytial Virus (RSV) Vaccine

CDC, ACIP, and the European Commission recommend the novel bivalent fusion F protein vaccine [31] and prefusion F protein vaccine [32] for adults aged $ $60. Populations at highest risk for severe RSV disease include those with chronic cardiac or lung disease, immunocompromised individuals, and nursing home residents. CDC estimates RSV causes 60,000-160,000 hospitalizations and 6,000-10,000 deaths annually in older adults [33].

3.6 Inhaled Therapy Management

GOLD 2024 merged "Issues Related to Inhaled Therapy" from former Chapter 3 with "Management of Inhaled Therapy" from former Chapter 4 into the current Chapter 3' s "Management of Inhaled Therapy," further divided into "Ability to Use Delivery Systems Correctly" and "Selection of Inhaler Devices." Most COPD medications are inhaled, making correct inhaler use critical for optimizing benefit-risk ratio. This requires appropriate device selection, education, regular technique checks, and re-education and device adjustment when necessary.

3.6.1 Ability to Use Delivery Systems Correctly Over two-thirds of patients make at least one error when using inhaler devices. While error types and frequencies vary by device characteristics, all devices require explanation, demonstration, and regular technique checks. Major errors involve inspiratory flow, duration, coordination, dose preparation, pre-inhalation exhalation, and post-inhalation breath-hold. The "teach-back" method (asking patients

to demonstrate device use) is more effective. Interventions guided by pharmacists, physicians, physiotherapists, nurses, and non-professional health coaches improve inhaler technique and adherence.

3.6.2 Selection of Inhaler Devices If patients are using their current device correctly, new treatments should ideally use the same device. If patients cannot use their current device correctly or no suitable medication is available in the same device, a systematic process should select a new device ensuring patient usability. Device selection depends on drug availability, device characteristics, patient acceptability and preference, and healthcare professional knowledge of correct use, with final decisions made through shared decision-making. Healthcare professionals must provide appropriate education including physical demonstration, video or technique demonstration, and on-site verification of mastery. Regular technique checks (preferably at each visit) are essential. Clinical lack of placebo devices often limits quality technique instruction. Encouraging patients to bring their personal devices to clinic is an effective option.

3.7 Pharmacotherapy for Smoking Cessation

Smoking cessation pharmacotherapy includes control medications for long-term abstinence (nicotine patches, bupropion, varenicline) and rapid-relief drugs for acute withdrawal symptoms (short-acting nicotine).

3.7.1 Nicotine Replacement Products Nicotine replacement therapy (gum, inhaler, nasal spray, transdermal patch, sublingual tablet, lozenge) effectively improves long-term quit rates and is more effective than placebo. Local irritation at administration sites is common, and non-ischemic chest pain and palpitations may occur. Medical contraindications include recent myocardial infarction or stroke. The safety of nicotine replacement after acute coronary syndrome is unclear, but data suggest waiting two weeks after cardiovascular events [34]. Continuous chewing of nicotine gum leads to swallowed secretions with minimal absorption and may cause nausea.

3.7.2 Electronic Cigarettes E-cigarettes are an effective nicotine replacement therapy for smoking cessation, though their effectiveness remains controversial. The long-term health effects of e-cigarettes, particularly in high-risk populations like COPD patients, are largely unknown.

3.7.3 Medications A meta-analysis of pharmacologic control treatments for COPD smokers (nicotine replacement therapy, bupropion, nortriptyline, varenicline) showed all medication groups (except nortriptyline) increased quit probability compared with placebo [29]. Long-term quit rates were 14%-27% with medications versus 5%-9% with placebo [35]. Another study showed higher continuous abstinence rates at weeks 9-24 with varenicline (58.3%) and bupropion (55.6%) compared with nicotine patch (38.2%) [36]. Varenicline and bupropion

showed similar efficacy, though varenicline patients smoked more cigarettes daily [36].

4. COPD Exacerbation Management

Key points: (1) COPD exacerbation is defined as an event within 14 days characterized by worsening dyspnea and/or cough and sputum. Exacerbations associate with increased local and systemic inflammation from airway infections, air pollution, or other lung injuries. (2) Since these symptoms are not COPD-specific, differential diagnoses must be considered, especially pneumonia, congestive heart failure, and pulmonary embolism. (3) Exacerbation management aims to minimize current impact and prevent subsequent events. (4) Short-acting $ $2-agonists, with or without short-acting anticholinergics, are recommended initial treatment. (5) Long-acting bronchodilator maintenance therapy should be initiated promptly; ICS should be considered in addition to dual bronchodilation in frequent exacerbators with elevated blood eosinophils. (6) Systemic corticosteroids improve lung function (FEV1) and oxygenation and shorten recovery (including hospitalization) in severe exacerbations, typically for $ $5 days. (7) Antibiotics may be used when indicated to shorten recovery, reduce early relapse risk, decrease treatment failure, and shorten hospitalization, typically for 5 days. (8) Methylxanthines are not recommended due to increased adverse effects. (9) Non-invasive ventilation is preferred for acute respiratory failure without absolute contraindications, as it improves gas exchange, reduces work of breathing, decreases intubation, shortens hospital stay, and improves survival. (10) Recovery time varies, typically requiring 4-6 weeks, with some patients not returning to pre-exacerbation functional status. After an exacerbation, preventive measures should be implemented.

Update points: GOLD 2024 is essentially consistent with GOLD 2023. The flowchart for exacerbation severity assessment and differential diagnosis is shown in [Figure 5: see original paper].

5.1.1 Key Points

COPD commonly coexists with other diseases significantly affecting prognosis. Comorbidities should not alter COPD management and should be treated per standard guidelines independent of COPD. Cardiovascular disease is a common and important comorbidity. Lung cancer is common in COPD patients and a leading cause of death. Annual LDCT screening for lung cancer is recommended in COPD populations caused by smoking; insufficient data exist to recommend annual LDCT in non-smoking-related COPD. Osteoporosis and depression/anxiety are common, important, often missed comorbidities associated with poor health status and outcomes. GERD associates with increased exacerbation risk and poor health status. When COPD is part of multiple disease management plans, treatment simplicity should be ensured and polypharmacy minimized.

5.1.2 Update Points

Compared with GOLD 2023, GOLD 2024 added several studies on three comorbidities: cardiovascular disease, obstructive sleep apnea with insomnia, and frailty.

5.2 Cardiovascular Disease

A large study in COPD patients without cardiovascular disease history found 25% increased risk of major adverse cardiac events (acute myocardial infarction, stroke, or cardiovascular death) [37]. This retrospective study using Ontario, Canada health administrative, pharmaceutical, laboratory, EMR, and other data followed patients with and without COPD from 2008-2016, comparing cardiac risk factors and comorbidities. Among approximately 5.8 million Ontarians aged $ $40 without cardiovascular disease history, 152,125 had COPD. After adjustment, COPD patients had 25% higher major adverse cardiovascular event rates (OR=1.25, 95%CI=1.23-1.27).

5.3 Obstructive Sleep Apnea and Insomnia

Insomnia in COPD patients correlates with higher outpatient visit and hospitalization rates [38]. Using Veterans Health Administration data from fiscal years 2012-2017, a retrospective cohort of COPD veterans found that among 1,011,646 COPD patients, 407,363 (38.8%) had insomnia. After adjustment, insomnia associated with higher outpatient and hospitalization rates, corticosteroid and/or antibiotic use, longer hospital stays, and increased 12-month hospitalization costs. These findings emphasize the importance of treating insomnia to reduce COPD burden on patients and healthcare systems.

5.4 Frailty

A meta-analysis showed frailty and pre-frailty associate with all-cause mortality, exacerbations, and hospitalization in COPD patients [39]. Conducted by a team from Ganyu District People' s Hospital in Lianyungang, China, the study included 10 studies with 13,203 COPD patients, showing frailty prevalence of 6.0%-51.0%. Compared with non-frail status, pre-frailty and frailty had hazard ratios for all-cause mortality of 1.48 (95%CI=0.92-2.40) and 2.64 (95%CI=1.74-4.02), respectively. Pre-frailty OR for all-cause hospitalization was 1.35 (95%CI=1.05-1.74) and frailty OR was 1.65 (95%CI=1.05-2.61). Frailty significantly predicted exacerbations in stable COPD (OR=2.20, 95%CI=1.26-3.81) but not moderate-to-severe exacerbations (OR=1.42, 95%CI=0.94-2.17). All-cause hospitalization results were not robust in sensitivity analysis, but frailty remained a significant predictor of all-cause mortality after adjusting for common confounders. Assessing frailty in COPD patients can facilitate secondary prevention and early intervention.

The European Respiratory Society published a review on frailty treatment in adults with chronic lung disease, including clinical management through geri-

atric care, rehabilitation, nutrition, pharmacotherapy, and psychotherapy [40]. Frailty is a complex syndrome characterized by loss of physiological reserve increasing vulnerability to adverse health outcomes. Most frailty knowledge derives from geriatric medicine, but its importance as a treatable trait in chronic respiratory diseases (including asthma, COPD, and interstitial lung disease) is increasingly recognized. Better understanding of frailty' s impact is prerequisite for optimizing clinical management.

6.1.1 Key Points

COPD patients with new or worsening respiratory symptoms, fever, and/or other possible COVID-19 symptoms, even if mild, should be tested for SARS-CoV-2 infection. Patients should continue oral and inhaled COPD medications as prescribed. During high community COVID-19 prevalence, spirometry should be limited to urgent cases or when essential for diagnosis or pre-procedure assessment. Social distancing and protection or appropriate isolation should not cause social isolation and inactivity; patients should maintain contact with friends/family via remote communication and remain active, ensuring adequate medication supply. Patients should be encouraged to use authoritative resources for COVID-19 information. Remote (phone/virtual/online) follow-up should be provided with printed checklists.

6.1.2 Update Points

For pharmacological treatment in COPD patients with suspected or confirmed COVID-19: nebulization can be used if indicated, along with antiviral drugs, corticosteroids, and immunomodulatory therapy. Current studies lack COPD subgroup data, but recommend COPD patients with COVID-19 receive standard treatment including antivirals, corticosteroids, IL-6 receptor blockers, and baricitinib.

6.2 Pharmacological Treatment for COPD in COVID-19 Patients

When providing COPD pharmacotherapy to patients with suspected or confirmed COVID-19, maintain existing COPD treatment and ensure adequate drug supply. Apply antibiotics and oral corticosteroids consistent with exacerbation treatment recommendations. GOLD 2024 adds that nebulization can be used with personal protective equipment when necessary.

ICS reduce exacerbation risk in COPD patients with exacerbation history but increase pneumonia risk. Laboratory studies show corticosteroids reduce antiviral interferon (Type I and III) production, increasing rhinovirus and influenza replication [41]. ICS impairs innate and acquired antiviral immune responses, causing delayed viral clearance, increased mucus secretion, impaired antimicrobial peptide secretion, and increased bacterial load during viral exacerbations [42]. Laboratory data show pretreatment of primary human nasal and airway epithelial cells with glycopyrrolate or formoterol reduces viral RNA levels and/or

titers, HCoV-229E receptor CD-13 expression, HCoV-229E RNA entry into acidic endosomes, and infection-induced cytokine production [including interleukin (IL)-6, IL-8, and interferon (IFN)-β]. Pretreatment with triple therapy (glycopyrrolate, formoterol, and budesonide) had additive inhibitory effects on viral titers and cytokine production, suggesting potential protective effects of ICS against COVID-19 not yet validated clinically [43]. No conclusive data support changing COPD maintenance therapy to reduce COVID-19 risk or due to concerns that therapy may increase COVID-19 risk. Similarly, no data exist on COPD patients using long-acting bronchodilators, LAMA or LABA, roflumist, macrolides, and SARS-CoV-2 infection outcomes/risk. Therefore, unless evidence supports otherwise, patients should continue COPD medications.

Nebulization increases aerosol generation and disease transmission risk; SARS-CoV-2 can survive in aerosols for up to 3 hours [44]. Studies report SARS-CoV-2 transmission to healthcare workers exposed to hospitalized COVID-19 patients receiving nebulization [45]. Therefore, healthcare workers should wear personal protective equipment (PPE) when providing nebulization therapy.

6.3 COVID-19 Treatment for COPD Patients with COVID-19

When providing COVID-19 treatment to COPD patients with suspected or confirmed infection, antiviral drugs, corticosteroids, and immunomodulators can be used. Randomized clinical trials for COVID-19 treatment focus on antiviral and anti-inflammatory therapies. Some drugs show positive results in hospitalized patients with severe COVID-19, including systemic corticosteroids. Current studies lack COPD subgroup data and analyses. COPD patients with COVID-19 should receive the same standard treatment as other patients, including antivirals, corticosteroids, IL-6 receptor blockers, and baricitinib.

7. "China Voice"

Notably, among the 148 new references in GOLD 2024, ten are from Chinese research teams, representing a "China Voice" on the international platform. These ten articles cover COPD epidemiology, diagnosis, treatment, and provide strong evidence supporting GOLD 2024 updates. Professor Chen Yanfan' s team used 2019 GBD data to show chronic respiratory diseases remain leading causes of global prevalence, mortality, and disability-adjusted life years (DALYs). Since 1990, absolute numbers have increased while some age-standardized estimates have decreased [46]. Professor Wang Zhaojun' s team similarly used 2019 GBD data to find that health inequality from socioeconomic status disparities is worsening, with increasing global wealth concentration potentially exacerbating COPD-related health disparities [47]. Professor Cao Chao' s team found secondhand smoke exposure associates with COPD risk, particularly in individuals with long-term exposure [48]. Academician Zhong Nanshan' s team found the COPD Assessment to Identify Unrecognized Respiratory Disease and Exacerbation Risk (CAPTURE) questionnaire sensitively identifies COPD patients requiring intervention for worsening symptoms or exacerbation/hospitalization

risk and can exclude those not needing treatment [49]. Professor Fan Yu' s meta-analysis showed frailty in COPD patients strongly correlates with all-cause mortality, exacerbations, and hospitalization risk [39]. Professor Yang Kehu' s team evaluated e-cigarette and nicotine replacement therapy effectiveness and safety for smoking cessation, finding e-cigarettes superior for 6-month continuous abstinence and 7-day point prevalence abstinence [35]. Professor Li You' s team examined RSV-associated hospitalization and mortality burden across age spectra, finding laboratory-based surveillance may severely underreport RSV disease burden in 5-year age groups [33]. Professor Lin Peijin' s team reviewed early pulmonary rehabilitation effects in hospitalized COPD exacerbation patients, finding early rehabilitation significantly reduces readmission rates [50]. Professor Zhang Xiaohui' s team reviewed different interventions for COPD smoking cessation, finding combined behavioral and pharmacotherapy most effective, with researchers needing to focus more on pharmacotherapy safety [51]. Professor Wu Xianbo' s team explored PRISm and cardiovascular outcomes in a large prospective cohort, finding PRISM individuals have higher cardiovascular disease risk, while those transitioning from PRISm to normal results show similar risk as those with normal lung function [52]. We thank these Chinese experts and scholars for their invaluable contributions to advancing COPD "prevention, promotion, diagnosis, control, treatment, and rehabilitation" !

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GOLD 2024 made substantial structural adjustments compared with GOLD 2023, with major updates concentrated in the first three chapters. The former Chapters 3 and 4 were merged into "Prevention and Management of COPD," with corresponding supplements and revisions focusing on smoking cessation, vaccination, and inhaled therapy. The smoking cessation section added pharmacotherapy details and evidence support, emphasizing cautious attitudes toward e-cigarette use. Vaccination recommendations align with current CDC guidelines, adding RSV vaccine guidance. The inhaled therapy section was greatly expanded and divided into two parts on teaching correct device use and device selection, reflecting GOLD 2024' s emphasis on ensuring proper inhalation. Additionally, GOLD 2024 updated COPD definition, diagnosis, and assessment. Following PRISm' s first inclusion in GOLD 2023, GOLD 2024 further expands the concept with updated prevalence data and emphasizes management of this population. Second, GOLD 2024 adds lung hyperinflation content, addressing previous knowledge gaps about this pathophysiological change. Regarding

lung function, it breaks from the fixed perception of post-bronchodilator indices, proposing pre-bronchodilator spirometry in clinical practice to improve COPD detection. Furthermore, to improve screening efficiency, GOLD 2024 recommends maximizing LDCT opportunities to screen for COPD, including using lung cancer imaging and incidental chest imaging abnormalities. Previous GOLD guidelines repeatedly emphasized blood eosinophil abnormalities; GOLD 2024 adds supporting clinical evidence. Finally, in imaging, GOLD 2024 adds interstitial lung abnormalities content, emphasizing clinical evaluation and follow-up for these patients. These updates demonstrate GOLD 2024' s substantial focus on improving COPD screening rates, pointing the way for future prevention and management.

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Note: Figure translations are in progress. See original paper for figures.