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Clinical Utility of the DECAF Score in Predicting In-Hospital Mortality and Adverse Outcomes in Acute Exacerbations of Chronic Obstructive Pulmonary Disease: A Narrative Review

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ABSTRACT:

Acute exacerbations of chronic obstructive pulmonary disease (AECOPD) are a leading cause of hospital admissions and are associated with substantial in-hospital mortality. Early risk stratification is essential to guide clinical decision-making, optimize resource utilization, and improve outcomes. Several prognostic tools have been evaluated in AECOPD; however, many lack disease specificity or practical bedside applicability. The Dyspnea, Eosinopenia, Consolidation, Acidemia, and Atrial Fibrillation (DECAF) score has emerged as a simple, COPD-specific prognostic model designed to predict in-hospital mortality. This narrative review synthesizes evidence from multiple observational studies, validation cohorts, and comparative analyses assessing the performance of the DECAF score in hospitalized AECOPD patients. Available data consistently demonstrate that the DECAF score provides good discriminatory ability for mortality prediction and often outperforms commonly used generic severity scores. Its ease of use and reliance on routinely available clinical parameters make it a valuable tool for early risk assessment and clinical triage in AECOPD.

Introduction

Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity and mortality worldwide. In 2021 it caused 3.5 million deaths, accounting for about 5 % of global mortality. The burden is especially severe in low- and middle-income countries where ~90 % of COPD deaths occur [1]. COPD exacerbations are characterized by a sudden worsening of respiratory symptoms that exceed day-to-day variation [2]. These acute exacerbations (AECOPD) contribute disproportionately to the disease burden, accounting for up to 12.5 % of hospital admissions and causing in-hospital mortality rates from 4.4 % to 25 %. Survivors have high rates of readmission (25 – 55 %) within a year.

COPD exacerbations result from complex interactions between airway inflammation, infection and systemic comorbidities. Pathophysiological changes include progressive airflow obstruction, hyperinflation, impaired gas exchange and respiratory muscle fatigue [3]. Systemic inflammation and oxidative stress also exacerbate comorbid conditions, particularly cardiovascular disease. Given this heterogeneity, early risk stratification tools are essential to guide management decisions, triage patients to appropriate care levels and allocate scarce resources. Generic severity scores—such as CURB-65, APACHE II and BAP-65—provide some prognostic information but were developed for pneumonia or general critical illness and may not capture COPD-specific risk factors. The DECAF score (Dyspnea, Eosinopenia, Consolidation, Acidaemia, Atrial Fibrillation) is a disease-specific tool derived in 2012 and validated in multiple cohorts. This review synthesises current evidence on the DECAF score for predicting in-hospital mortality in AECOPD, compares it with other prognostic models and discusses clinical implications and future directions.

Journal of Chemical Health Risks

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Pathophysiological Basis of Mortality in AECOPD

COPD is a chronic inflammatory disease characterized by irreversible airflow limitation. Persistent exposure to noxious particles (e.g., tobacco, biomass smoke or air pollution) injures airway epithelium, induces mucous gland hyperplasia and leads to destruction of alveolar septa and elastic fibres. Loss of elastic recoil causes dynamic hyperinflation, increasing intrathoracic pressure and placing respiratory muscles at a mechanical disadvantage [4]. Acute exacerbations—often precipitated by viral or bacterial infections—worsen airway inflammation and promote ventilation-perfusion mismatch, resulting in hypoxemia, hypercapnia and respiratory acidosis [5]. Dynamic hyperinflation increases intrinsic positive end-expiratory pressure (PEEP_i), elevating intrathoracic pressure and reducing venous return; this contributes to hypotension and cardiac dysfunction during severe exacerbations [6]. Systemic inflammation, oxidative stress and sympathetic activation further stress the cardiovascular system. These pathophysiological processes underlie the components of prognostic scores.

Comorbidities amplify mortality risk. Cardiovascular disease (CVD) is particularly prevalent among COPD patients and shares risk factors such as smoking, ageing and air pollution. Epidemiological studies show COPD increases the risk of CVD events (odds ratio ≈ 2.46) and that the frequency and severity of exacerbations correlate with myocardial infarction and pulmonary embolism. A prospective study in China found cardiovascular comorbidity increased the risk of AECOPD by ≈4-fold; multivariable analysis identified CVD, higher dyspnea score (mMRC ≥ 3) and irregular follow-up as significant risk factors [7]. Other comorbidities—including chronic heart failure, diabetes, malnutrition and anaemia—contribute to mortality. Therefore, prognostic models must account for both pulmonary and systemic factors.

Overview of Prognostic Assessment Tools in AECOPD

CURB-65 and APACHE II

The CURB-65 score was originally designed for community-acquired pneumonia. It assigns one point each for confusion, blood urea >7 mmol/L, respiratory rate ≥30 min^{-1}, low blood pressure and age ≥65 years. A retrospective cohort of 1 583 AECOPD patients reported that mortality increased markedly with increasing CURB-65 score, with an area-under-the-curve (AUC) of 0.882 (sensitivity 94 %, specificity 67.2 %) for predicting in-hospital death. However, CURB-65 focuses on infection-related parameters and may underestimate COPD-specific risks.

APACHE II evaluates acute physiology, age and chronic health variables. In the same cohort, APACHE II showed excellent discrimination (AUC 0.965, sensitivity 91.6 %, specificity 89.2 %) for mortality prediction [8]. Its complexity and requirement for multiple laboratory values limit routine use outside intensive care.

BAP-65

The BAP-65 score incorporates blood urea nitrogen ≥25 mg/dL, altered mental status (Glasgow Coma Scale < 14), pulse ≥109 min^{-1} and age ≥65 years. Risk classes correlate with the need for mechanical ventilation and in-hospital mortality; high-risk (class V) patients require close monitoring. In a prospective Indian cohort, mean BAP-65 class among survivors was 1.92 ± 1 compared with 4.28 ± 0.91 in those who died, yielding 71 % sensitivity and 94 % specificity.

Other Scores

Several other tools have been proposed. The CAPS and NEWS scores incorporate physiological parameters but have variable performance. The CAUDA70 score adds confusion, acidosis, urea, dyspnea, albumin and age factors. In an Indian study of 100 patients, mortality rates were significantly associated with BAP-65, DECAF and CAUDA70 scores; 29.2 % of patients with BAP-65 >3 and 37.5 % with CAUDA70 >2 died. However, the study concluded all three scores help stratify risk and are easy to apply. A 2024 cross-sectional study comparing DECAF, modified DECAF and BAP-65 found that BAP-65 had greater specificity and higher AUC than the other scores [9].

Despite these advances, generic scores may not fully capture COPD-specific pathophysiology. For example, dyspnea severity and eosinopenia—strong predictors of COPD mortality—are absent from generic tools. The DECAF score was developed to address this gap.

Journal of Chemical Health Risks

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Components and Interpretation of the DECAF Score

The DECAF score comprises five variables:

1. **Dyspnea severity**: quantified using the extended Medical Research Council (eMRCD) scale. Stable-state dyspnea was the strongest predictor of mortality in the derivation study. Scores of 5a/5b confer two points; scores 4–5b represent high symptom burden [10].
2. **Eosinopenia**: peripheral blood eosinophil count $<0.05 \times 10^9$/L (eosinophils <50 µL) indicates immune suppression or systemic stress. Eosinopenia has been linked to increased infection severity and mortality.
3. **Consolidation**: radiographic evidence of pneumonia; consolidation on chest radiography signals severe infection and is associated with poor outcome.
4. **Acidaemia**: arterial pH <7.30 or <7.35 in presence of hypercapnia. Acidosis reflects ventilatory failure and is an established mortality predictor.
5. **Atrial Fibrillation (AF)**: baseline or new-onset AF; arrhythmias increase hemodynamic instability and mortality.

Each variable scores one point (except severe dyspnea which may score two). Total scores range from 0–6. The original validation study stratified patients into low risk (0–1), intermediate risk (2) and high risk (≥3), recommending early discharge for low-risk patients and escalation of care for high-risk groups. The DECAF score is simple to calculate from routinely available data, does not require complex laboratory tests and can be assessed within the first 24 hours of admission.

Evidence Supporting the DECAF Score

Derivation and Validation

Steer and colleagues derived the DECAF score from a multicentre cohort of 920 patients and validated it in an external cohort of 791 patients. The score achieved AUC 0.83 (internal) and 0.82 (external), outperforming CURB-65, BAP-65, APACHE II and CAPS scores [11]. Importantly, DECAF identified low-risk patients (scores 0–1) with in-hospital mortality around 1 % and high-risk patients (scores 3–6) with mortality >35 %, thereby guiding discharge and palliative decisions [12].

International Validation and Meta-analysis

A multicentre UAE study (2019–2021) including 530 patients confirmed the DECAF score's prognostic value: AUC 0.80 for inpatient mortality and readmission. Mean hospital length of stay increased dramatically with higher DECAF scores: 3.6 ± 2 days for score 0 versus 29.8 ± 31.4 days for score 6. A meta-analysis of 17 studies (8 329 patients) reported pooled sensitivity and specificity of 0.76 and 0.76 respectively, with an overall AUC of ~0.82. Subgroup analysis showed best discrimination at a cutoff of ≥3 (AUC 0.83). DECAF outperformed CURB-65, BAP-65, APACHE II and CAPS across studies [13].

A cross-sectional Indian study of 200 emergency department patients (2023) found DECAF to have the highest AUC (0.80) compared with BAP-65, NEWS, CURB-65 and qSOFA. DECAF's odds ratio for in-hospital mortality was 2.8, and for mechanical ventilation 2.3; sensitivity and specificity were 85 % and 75 % respectively. These findings support using DECAF as the preferred prognostic tool in resource-limited settings [14]. Another rural Indian study (100 patients) reported that DECAF and BAP-65 had comparable AUCs for mortality (0.83 vs. 0.79) and mechanical ventilation (0.77), underscoring the practicality of both scores.

Modified DECAF

Given the influence of dyspnea on the original score, a modified DECAF (m-DECAF) replaced stable dyspnea with acute dyspnea severity. In a 2025 prospective study of 120 patients, the m-DECAF score distinguished low- and intermediate-risk groups with 100 % survival, while the high-risk group had 75.86 % mortality and required invasive ventilation in 89.65 % of cases; average hospital stay was 17.43 days [14]. A separate cross-sectional analysis of 51 patients found strong correlation between DECAF and m-DECAF (Spearman's $\rho = 0.702$) but little evidence that m-DECAF improved outcomes [15].

Other Comparative Studies

A Turkish cohort compared DECAF with the Ottawa COPD Risk Score and found both scores useful; using a

Journal of Chemical Health Risks
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JCHR (2026) 16(1), 1796-1802 | ISSN:2251-6727

cutoff of 3, DECAF had 63 % sensitivity and 78 % specificity with AUC 0.762 [16]. A Swiss study compared BAP-65, modified DECAF, NEWS and a new AECOPD-COMBI score; BAP-65 performed best (AUC 0.79), modified DECAF had AUC 0.72, while the new COMBI score achieved AUC 0.9 but required complex calculations [17].

Collectively, these studies demonstrate robust validation of the DECAF score across diverse settings, with consistent discriminative performance and practical application.

Comparative Performance and Emerging Models

New Predictive Scores

Recognizing limitations in existing scores for severe AECOPD, Hu et al. (2024) developed a novel five-variable score comprising age >80 years, confusion, lymphopenia, chronic heart failure and leukocytosis. In the validation cohort, the new score achieved AUC 0.826, outperforming DECAF (0.783), BAP-65 (0.73) and CURB-65 (0.652) [18]. The new score's sensitivity was 69.2 % and specificity 81.2 % [19]. Age, confusion, low lymphocytes and high white cell counts were identified as independent predictors of death.

Machine-Learning Models

Machine-learning (ML) approaches may enhance predictive accuracy. A 2025 multicentre Chinese study used echocardiographic and clinical variables to train 11 ML algorithms. The best model—Light Gradient Boosting Machine (LightGBM)—produced an AUC 0.956, accuracy 92.1 %, sensitivity 0.891 and specificity 0.933. The model used nine features after feature selection and was converted into a web-based tool for clinician use [20]. While promising, such models require external validation and transparency to ensure clinical trust.

DECAF-L and Lactate Integration

Hypoxemia during severe AECOPD results in anaerobic metabolism and elevated serum lactate. A 2025 editorial from the Eurasian Journal of Emergency Medicine summarised a prospective study where adding lactate to the DECAF score (DECAF-L) improved prognostic accuracy. BAP-65 and DECAF were associated with patient disposition (discharge vs. ward vs. ICU), but DECAF-L emerged as the only independent predictor of 30-day mortality, with an odds ratio of 1.296 per unit increase. Integration of lactate, a readily available biomarker, may refine early risk stratification in emergency departments.

Systematic Reviews and Meta-analyses

A 2024 systematic review evaluated 53 prognostic models across 46 studies. Most models used logistic regression, with median sample size 672 and median of five predictors. The pooled AUC was 0.80 for mortality and 0.84 for hospitalization outcomes. However, 52 models had a high risk of bias, mainly due to small sample sizes, reliance on univariate predictor selection and inadequate validation. Only the PEARL score achieved low risk of bias. The authors called for standardized development and validation procedures and broader external validation.

Biomarkers, Eosinophils and Other Risk Factors

Eosinophil Counts and Stability

Eosinopenia is an integral component of the DECAF score. In a cohort of 82 patients with AECOPD and community-acquired pneumonia, eosinopenia (<50 μL) was associated with higher infection biomarkers and markedly lower 18-month survival [21]. A large 2025 study examined eosinophil stability during hospitalization. Patients were classified into persistent high, decreased, increased and persistent low eosinophil (EOS) groups. The persistent high EOS group had the lowest rates of ICU transfer (2.9 %), invasive mechanical ventilation (1.7 %) and in-hospital mortality (0.2 %). In contrast, the persistent low EOS group had higher hospital costs and mortality (4.5 %). Multivariate analysis showed that persistent high eosinophil counts were protective (adjusted OR 0.77, 95 % CI 0.59–0.99), whereas prior exacerbations and higher Charlson index increased risk. These findings support the prognostic role of eosinophil counts beyond a single measurement.

Inflammatory Biomarkers

Numerous studies link systemic inflammatory indices to mortality. A retrospective analysis of 104 ICU patients with severe COPD exacerbation compared survivors and non-survivors. Non-survivors had significantly higher neutrophil-to-lymphocyte ratio (NLR) (23.0 vs. 8.7) and systemic immune-inflammation index (SII). Age, C-reactive protein (CRP), NLR,

Journal of Chemical Health Risks
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monocyte-to-lymphocyte ratio and SII positively correlated with mortality, whereas higher lymphocytes were protective. Incorporating these biomarkers into risk models may improve discrimination.

Cardiovascular and Other Risk Factors

Age, heart failure and baseline functional status are important prognostic determinants. The new predictive score highlighted the impact of age >80 years, confusion, lymphopenia, chronic heart failure and leukocytosis on mortality [22]. Machine-learning models identified echocardiographic features among key predictors, emphasising the importance of assessing cardiac function in AECOPD. Furthermore, a case-control study in China found cardiovascular disease, high mMRC dyspnea scores and irregular follow-up as risk factors for exacerbations. Observational data also link smoking history, anemia, low body mass index, renal dysfunction and malnutrition to poor outcomes.

Clinical Implications

Effective triage is critical for reducing mortality and optimizing resource utilization. The DECAF score stratifies patients into risk categories soon after admission using variables that are routinely collected. Low-risk patients (0–1) have mortality <2 % and may be candidates for early discharge or treatment in general wards [17]. High-risk patients (≥3) have mortality >35 % and should receive closer monitoring, early escalation of care and consideration of ventilatory support. Incorporating DECAF into emergency department protocols can aid decisions regarding ward vs. ICU admission, non-invasive vs. invasive ventilation and need for palliative discussions. Evidence from India demonstrates that DECAF has the highest sensitivity and specificity among available scores and remains practical in resource-limited settings.

The addition of biomarkers may further refine risk stratification. The DECAF-L score—including serum lactate—provided superior prediction of 30-day mortality (odds ratio 1.296 per unit increase), suggesting that lactate measurement at presentation could guide early decisions. Machine-learning models offer even greater accuracy but require electronic health record integration, interpretability and external validation. Meanwhile, ongoing evaluation of eosinophil stability, NLR, CRP and other biomarkers may help clinicians identify patients at risk of deterioration. Tools like the new predictive score by Hu et al. or the AECOPD-COMBI score may supplement DECAF for severe exacerbations.

Limitations and Future Research

Despite strong evidence supporting the DECAF score, several limitations must be acknowledged. Most validation studies are observational and subject to confounding. Many were conducted in single centres or specific regions, potentially limiting generalizability. The 2024 systematic review found that 52 of 53 prognostic models had a high risk of bias due to small sample sizes, univariate predictor selection and inadequate validation. Standardization of model development, reporting and validation—using protocols such as TRIPOD and PROBAST—is necessary to ensure reliability. In addition, cut-off values for DECAF components (e.g., eosinophil counts) may vary across populations; further work should define optimal thresholds for different ethnic groups and healthcare settings.

Emerging tools like DECAF-L and ML-based models show promise but require large multi-centre studies and user-friendly interfaces. ML models must also be explainable and incorporate dynamic clinical parameters. Integration of echocardiographic data, patient-reported outcomes, comorbidity indices and biomarker trajectories could enhance prediction. Research should also explore how implementing these tools alters clinical pathways, hospital length of stay and cost-effectiveness. Finally, models must be validated in primary care and outpatient settings to identify patients at risk of impending exacerbations before hospitalization.

Conclusion

COPD exacerbations remain a leading cause of hospitalization and death worldwide. The DECAF score provides a simple, bedside tool that reliably predicts in-hospital mortality and guides clinical decision-making. Its components—dyspnea severity, eosinopenia, consolidation, acidaemia and atrial fibrillation—capture key pathophysiological processes and comorbidities unique to COPD. Extensive validation across diverse populations demonstrates robust discriminatory performance, with pooled AUC around 0.82 and superior accuracy compared with generic

Journal of Chemical Health Risks

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JCHR (2026) 16(1), 1796-1802 | ISSN:2251-6727

scores. The DECAF score identifies low-risk patients suitable for early discharge and high-risk patients who need intensive monitoring or ventilatory support. Modifications (m-DECAF) and biomarkers (DECAF-L) may improve its predictive power, and machine-learning models offer further potential. However, careful validation and standardization are essential. Future research should focus on integrating biomarkers, echocardiographic data and patient-centred outcomes into scalable, explainable models to personalize care for patients with AECOPD.

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