CARDIO-PULSE: CARDIOvascular disease and Chronic Obstructive PULmonary DiseaSE: a real-world observational study in Australian primary care

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LIST OF ABBREVIATIONS

Abbreviation or special term	Explanation
ADEPT	Anonymised Data Ethics and Protocol Transparency committee
Aus CVD	Australian CVD risk score
CAC	Coronary artery calcium (CVD risk assessment score)
CI	Confidence interval
COPD	Chronic obstructive pulmonary disease
CV	Cardiovascular
CVD	Cardiovascular disease
ENCePP	European Network of Centres for Pharmacoepidemiology and Pharmacovigilance
ERS	European Respiratory Society
GOLD	Global Initiative for Chronic Obstructive Lung Disease
HR	Hazard ratio
ICRA	International Cardiovascular and Respiratory Alliance (ICRA)
ICS	Inhaled corticosteroids
ICPC	International Classification of Primary Care
IRR	Incidence rate ratio
FEV1	Forced expiratory volume in 1 second
ppFEV1	Percentage predicted FEV1
FVC	Forced vital capacity
LABA	Long-acting β_2 agonist
LAMA	Long-acting muscarinic agonist
MACE	Major adverse cardiovascular event
MESA	Multi-ethnic study of atherosclerosis
OPCA	Optimum Patient Care Australia
OPCRDA	Optimum Patient Care Research Database Australia
PCRS	Primary Care Respiratory Society
REG	Respiratory Effectiveness Group
SABA	Short-acting β_2 agonist
SAMA	Short-acting muscarinic agonist
TSANZ	Thoracic Society of Australia and New Zealand

1. BACKGROUND & RATIONALE

Chronic obstructive pulmonary disease (COPD) and cardiovascular disease (CVD) are both major global public health concerns [1] [2]. In 2021, COPD, ischaemic heart disease and stroke were in the top 10 leading causes of death worldwide and both COPD and stroke were in the top 20 leading level 3 causes of global years lived with disability [2].

Studies have shown that people with COPD are 2–5 times more likely to have CVD [3] and around 25% more likely to experience major adverse cardiovascular events (MACE) [4] compared to those without COPD. This is partly due to shared risk factors, such as ageing, smoking and socioeconomic deprivation, but also due to COPD-related systemic inflammation which can diminish peripheral blood flow and lead to CVD progression [5] [6]. Indeed, mortality due to CVD in people with COPD is higher than that due to respiratory failure [7]. However, knowledge about CVD incidence in people with COPD in the primary care setting is limited as most studies have been conducted in secondary care [8].

The most recent (2026) report from the Global Institute for Chronic Obstructive Lung Disease (GOLD) highlights the importance of identifying CVD in people with COPD [9]. Risk of acute cardiovascular (CV) events, such as myocardial infarction and stroke, are highest in people during a COPD exacerbation [10] [11] but this risk remains high up to 12 months post-exacerbation [10] [12] [13] [14] so it is important that routine monitoring in primary care is prioritised. Among all patients with COPD, assessment tools can help to identify CV risk without the need for invasive diagnostic measures. In Australia, the recommended CV risk score in primary care since July 2023 is the *AusCVDRisk* score [15] (**Table 1**), which has been recalibrated from New Zealand's PREDICT equations [16]. Prior to this, CVD risk scores were based on Framingham risk score but with additional risk criteria that were recalibrated for the Australian population; however, this risk score missed some key risk factors such as socioeconomic disadvantage [17]. Independent research suggests that the *AusCVDRisk* score performs well in the Australian primary care setting [18]. However, it is known that CVD risk scores tend to be underestimate risk of CVD in COPD patients [9]. CVD may also be difficult to identify in the COPD population as non-specific symptoms such as nocturnal cough and paroxysmal dyspnoea are common to both conditions.

Table 1: CVD risk scores in Australian primary care

Time period	Name of assessment	Target group	Features	Risk definitions
Recommended CVD risk assessments in primary care				
2012 to Jul 2023	Absolute CVD risk score (**ACVDR**) [17]	Inclusion criteria 45-74 yrs; 35-74yrs (first nations); <60yrs (diabetes) Exclusion criteria CVD Diabetes (>60yrs) Diabetes+microalbuminuria CKD SBP≥180/DBP≥110 Familial hypercholesterolaemia Serum total cholesterol≥7.5mmol/L	Based on Framingham risk score but with additional risk criteria - recalibrated for Australian population, but missing some key risk factors (e.g., socioeconomic disadvantage)	5-year risk defined as high: >15%; intermediate: 10 to 15%; low: <10%
Jul 2023 to present	**AusCVDRisk** [15]	Inclusion criteria 45-79 yrs; 35-79 yrs (diabetes); 30-79 yrs (first nations) Exclusion criteria CVD	Based on the New Zealand PREDICT algorithm recalibrated for Australian population. Special consideration for Aboriginal and Torres Strait Islander populations and socioeconomic disadvantage	5-year risk defined as high: ≥10%; intermediate: 5 to <10%; low: <5%
Other risk scores				
2009 to present	Coronary Artery Calcium (**CAC**) score [19]	Inclusion criteria 45-84yrs Not recommended as a population screening test	Identification of plaque in coronary arteries using CT scanners. Reference values based on the Multi-Ethnic Study of Atherosclerosis (MESA) study	Risk defined as: low: ≤10; mild-moderate: 11-100; moderate-high: 101 - 400; >400 high [10-year risk: >20%]

Once comorbid COPD-CVD has been identified, treatment and prevention strategies become a priority, but previous research has shown underuse of appropriate therapies in patients with comorbid disease [20] and there are concerns that the attending physician tends to focus on their own condition of interest (i.e., lung/heart) such that the second morbidity can go undertreated [9]. Management strategies should combine tailored approaches with multidisciplinary expertise in both lung and heart health to take account of the overlap between symptoms and drug-drug interactions. Bronchodilator treatments for COPD, such as long-acting beta-2 agonists (LABA) may increase heart rate [21] and short-acting antimuscarinic agonists (SAMA), such as ipratropium bromide, may increase the risk of heart failure [22], which means that long-acting muscarinic agonists (LAMAs)

tend to be preferred over LABAs for patients with comorbid COPD-CVD [22]. There is also some evidence that statins have a protective role for both conditions due to their broad anti-inflammatory properties [23]. Conversely, there are concerns regarding the impact of CVD treatments such as non-selective beta-blockers in COPD patients which may worsen bronchoconstriction [21].

As primary care is the first point-of-contact for most health concerns, regular monitoring of comorbid COPD-CVD patients is vital to determine efficacy of medications and adverse effects. Good quality management of both COPD and CVD involves assessment of adherence, appropriate use of prescribed treatments, and lifestyle risk factors (e.g., smoking). However, there has been limited research on how people with comorbid COPD-CVD are treated in primary care. We urgently need greater understanding of the relationship between COPD and CVD, including how risk assessments are used to guide treatment and prevention strategies.

2. AIMS & OBJECTIVES

The overarching aim of this study is to identify priority areas of action in relation to people with comorbid COPD-CVD and people with COPD at risk of developing CVD. This includies exploring the risk of CVD among high-risk[1] COPD patients and how CVD risk assessments can be used to optimise prevention and treatment strategies. **Table 2** shows the study aims, research questions and objectives.

[1] 'High risk' defined as ≥2 COPD exacerbations in the 24 months prior to the index date

Table 2: Study aims, research questions and objectives

Study Aim	Research Question	Study Objectives
1. Identify the risk of CVD events in patients with high-risk COPD compared to matched controls and to explore the predictors of future CVD events in high-risk COPD	1a) Do patients with high-risk COPD have a higher frequency of CVD events compared to matched controls without COPD?	1a) Calculate the incidence of new-onset CVD events in patients with high-risk COPD when compared to age-gender-smoking matched controls
	1b) What proportion of patients with high-risk COPD currently have a CVD risk score documented in Australian primary care (general practice) and, where available, are their risk scores higher than similar patients without COPD?	1b) Describe the use of CVD risk score assessments high-risk COPD, and compare this with age-gender-smoking matched controls 1c) Describe the distribution of CVD risk scores in patients with high-risk COPD
	1c) How do other factors influence the risk of future CVD events in patients with high-risk COPD?	1d) Investigate factors associated with receiving a CVD risk assessment in patients with high-risk COPD 1e) Investigate factors associated with future CVD events in patients with high-risk COPD
2. Identify the time to COPD maintenance therapy change in patients with COPD & CVD and whether it reduces future CV and respiratory events	2a) Following a COPD exacerbation, do patients with comorbid CVD have their maintenance therapy changed more quickly than those without – and does baseline therapy make a difference?	2a) Compare time from exacerbation to COPD maintenance therapy change in patients with and without a prior CVD diagnosis 2b) Compare time from exacerbation to COPD maintenance therapy change by CVD diagnosis and baseline maintenance therapy class and regimen
	2b) Following a COPD exacerbation, is maintenance therapy adjustment associated with reduced COPD exacerbations and CVD events?	2c) Assess the association of maintenance therapy change with the subsequent occurrence of CVD events in patients with and without a prior CVD diagnosis

3. STUDY DESIGN AND POPULATION

3.1. Data sources

We will carry out an observational cohort study using data from Australian primary care electronic medical records from the Optimum Patient Care Research Database Australia (OPCRDA) [24,25]. The OPCRDA is a real-world, longitudinal, research database that is maintained by Optimum Patient Care Australia (OPCA). It contains anonymised health data from over one million patients from primary care across Australia.

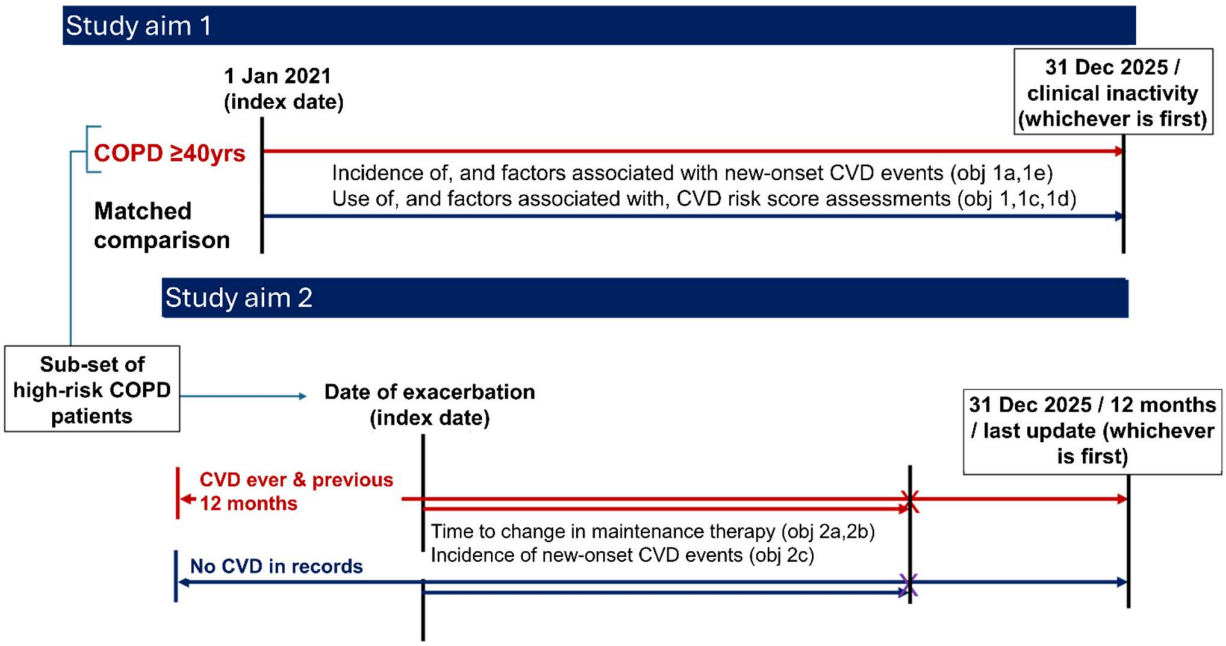
3.2. Study population and design

The population will comprise adults aged ≥40 years with COPD, defined as a documented diagnosis of COPD and a prescription for COPD medication in the previous 2 years, who are clinically active (prescription and/or consultation data) on the OPCRDA database from 1 January 2021. The design for this study is shown in **Figure 1**.

For the first study aim, people with COPD will be matched by age/gender/smoking status to a cohort of individuals without COPD. Index date will be 1 January 2021. Individuals will be followed up for 3–5 years and incidence of new-onset CVD events, CVD risk score assessments and absolute risk scores will be compared.

For the second study aim, the cohort will be restricted to individuals who are at high risk (i.e. have ≥2 exacerbations in the previous 24 months) and have at least one exacerbation within the follow-up period (up to 5 years to 31 December 2025). The first exacerbation will be taken as the index date. People with pre-existing CVD (ever – and in the previous 12 months – defined as stroke, myocardial infarction [MI], angina, peripheral arterial disease, atrial fibrillation, and/or heart failure) will be compared to those without CVD in relation to time to maintenance therapy change and incidence of new-onset CVD.

Figure 1: Study design of CARDIO-PULSE study



3.2. Eligibility criteria

Inclusion criteria

- Adults aged ≥40 years on 1 January 2021
- At least one consultation/therapy in the 12 months period prior to index date
- Diagnosis of COPD with at least one exacerbation (study 1)
- Meeting eligibility criteria for CVD risk score assessment (study 1; see **Table 1**)
- Diagnosis of high-risk[2] COPD (exposure arm; study 2)

Exclusion criteria

- No other chronic respiratory conditions, e.g. asthma

(a sensitivity analysis will be conducted to see the effect of including other chronic respiratory conditions, i.e. omitting the exclusion criteria)

[2] *'High risk' defined as ≥2 exacerbations in the 24 months prior to the index date

4. STUDY VARIABLES AND STUDY OUTCOME DEFINITIONS

4.1. New-onset CVD events (Aim 1; Objectives 1a, 1e)

Incidence of CVD events from index date (1 Jan 2020) will be evaluated (Objective 1a). Factors associated with new-onset CVD events (Objective 1e) will be investigated for: sociodemographics, clinical characteristics/indicators (including COPD exacerbations), comorbidities, and drug therapy classes, as outlined in **Table 3**.

Table 3: Variable list for CARDIO-PULSE study: Objectives 1a,1e

Variable	Collection of data
CVD event (outcomes)	
Heart failure	New-onset events at follow-up (≥3–5 years)
Stroke	New-onset events at follow-up (≥3–5 years)
Myocardial infarction	New-onset events at follow-up (≥3–5 years)
Angina	New-onset events at follow-up (≥3–5 years)
Peripheral arterial disease	New-onset events at follow-up (≥3–5 years)
Atrial fibrillation	New-onset events at follow-up (≥3–5 years)
Sociodemographics (exposure)	
Age	At index date
Sex	At index date
Ethnicity	At index date (note: proxy = nationality)
Calendar year	At index date
Lifestyle and lifestyle modification (exposure)	
Body mass index	Baseline (within 12 months of index date)
Smoking status	Baseline (within 12 months of index date)
Alcohol consumption	Baseline (within 12 months of index date)
Clinical measurements / indicators (exposure)	
Number of exacerbations	Baseline (within 12 months of index date)
Family history of CVD	Ever in patients' records
ppFEV1	Baseline (within 12 months of index date)
FVC	Baseline (within 12 months of index date)
Serum total cholesterol	Baseline (within 12 months of index date)
Systolic blood pressure	Baseline (within 12 months of index date)
Diastolic blood pressure	Baseline (within 12 months of index date)
Eosinophil count	Highest eosinophil count within 5 years of index date
CVD risk score	Baseline (within 12 months of index date)
Comorbidities (exposure)	
Diabetes	Ever in patients' records – and within 12 months of index
Chronic kidney disease	Ever in patients' records – and within 12 months of index

Coronary artery disease	Ever in patients' records – and within 12 months of index
Heart failure	Ever in patients' records – and within 12 months of index
Stroke	Ever in patients' records – and within 12 months of index
Angina	Ever in patients' records – and within 12 months of index
Myocardial infarction	Ever in patients' records – and within 12 months of index
Peripheral arterial disease	Ever in patients' records – and within 12 months of index
Atrial fibrillation	Ever in patients' records – and within 12 months of index
Sleep apnoea	Ever in patients' records – and within 12 months of index
Familial hypercholesterolaemia	Ever in patients' records – and within 12 months of index
COPD therapy class (exposure)	
LABA ± SABA	Yes/no at baseline (within 12 months of index date)
LAMA ± SABA	Yes/no at baseline (within 12 months of index date)
ICS/LABA ± SABA	Yes/no at baseline (within 12 months of index date)
LABA/LAMA ± SABA	Yes/no at baseline (within 12 months of index date)
ICS/LABA/LAMA ± SABA	Yes/no at baseline (within 12 months of index date)
Other	Yes/no at baseline (within 12 months of index date)

4.2. Use of, and factors associated with, CVD risk score assessments (Aim 1; Objectives 1b, 1c, 1d)

CVD risk score will be investigated as a yes/no variable (where available [Objectives 1b,1c]) and as absolute values (Objective 1d). Type of CVD risk score will also be investigated. The exposure measures defined in **Table 4** will be used to explore factors associated with CVD risk score assessments (Objective 1d).

Table 4: Variable list for CARDIO-PULSE study: Objectives 1b,1c,1d

Variable	Collection of data
CVD risk score (outcome)	
Framingham-based CVD risk score (prior to 2023) [yes/no and absolute values]	During follow-up (≥3–5 years)
Aus CVD risk score, recalibrated from New Zealand's PREDICT equations	During follow-up (≥3–5 years)
MESA risk score	During follow-up (≥3–5 years)
Coronary artery calcium (CAC) risk score	During follow-up (≥3–5 years)

4.3. Time-to-change in maintenance therapy (Aim 2: Objectives 2a,2b)

Time-to-change in COPD maintenance therapy during the 12-month period after a COPD exacerbation (Objective 2a) will be grouped into: (change to) LABA or LAMA; ICS/LABA ± SABA; LABA/LAMA ± SABA; ICS/LABA/LAMA ± SABA; and other (see **Table 5**). Information will be stratified by baseline information of COPD inhaler usage (Objective

2b) to assess whether/how individuals step-up/down to different therapies after having a COPD exacerbation.

Table 5: Variable list for CARDIO-PULSE study: Objectives 2a,2b

Variable	Collection of data
Therapy class (outcome)	
LABA monotherapy	Date of prescription (change only) during 12-month follow-up
LAMA monotherapy	Date of prescription (change only) during 12-month follow-up
ICS/LABA ± SABA	Date of prescription (change only) during 12-month follow-up
LABA/LAMA ± SABA	Date of prescription (change only) during 12-month follow-up
ICS/LABA/LAMA ± SABA	Date of prescription (change only) during 12-month follow-up
Other	Date of prescription (change only) during 12-month follow-up
Therapy class (exposure)	
LABA ± SABA	Yes/No at baseline (within 12 months of index date)
LAMA ± SABA	Yes/No at baseline (within 12 months of index date)
ICS/LABA ± SABA	Yes/No at baseline (within 12 months of index date)
LABA/LAMA ± SABA	Yes/No at baseline (within 12 months of index date)
ICS/LABA/LAMA ± SABA	Yes/No at baseline (within 12 months of index date)
Other	Yes/No at baseline (within 12 months of index date)
Other therapy changes	
Change in individual COPD therapies	Date of prescription (change only) during 12-month follow-up
Change to macrolides (erythromycin, azithromycin, other)	Date of prescription (change only) during 12-month follow-up

4.4. Incidence of new-onset CVD events (Aim 2; Objective 2c)

New-onset CVD events (Objective 2c) will be determined using a combination of freetext and diagnostic codes (SNOMED, PYEFINCH, DOCLE) and will be defined by heart failure, angina, peripheral vascular disease, myocardial infarction, and/or stroke (**Table 6**). As this involves incidence of new-onset events, we will only include the specific CVD event within ≥3 months of another event of the same type (i.e., likely to be duplicated events), but will allow two different events (e.g., stroke and heart failure) to occur at any point. The effect of time-to-change in maintenance therapy will be modelled as separate categories by month, also including “no therapy change” as a separate category.

Table 6: Variable list for CARDIO-PULSE study: Objective 2c

Variable	Collection of data
CVD event (outcome)	
Heart failure	New-onset event at 12 months follow-up
Stroke	New-onset event at 12 months follow-up
Myocardial infarction	New-onset event at 12 months follow-up

Angina	New-onset event at 12 months follow-up
Peripheral arterial disease	New-onset event at 12 months follow-up
Atrial fibrillation	New-onset event at 12 months follow-up
Time to change in COPD maintenance therapy (exposure)	
Months from exacerbation to change	During 12 months follow-up. No changes will be treated as a separate category.

5. STATISTICAL ANALYSIS

5.1. Statistical Analysis Plan

COPD vs no COPD: CVD events and CVD risk score assessments (Aim 1)

Baseline characteristics of the population for study population for the first aim will be described for each of the covariates and outcomes. People will enter on 1 Jan 2020 ('index date') and will be active in the cohort for at least 3 years; the follow-up period will be 3–5 years. The study population will comprise people with high-risk COPD. The comparison population will comprise age-gender-smoking-matched patients without COPD. We will use Poisson regression (or negative binomial if overdispersion is present) to calculate IRR (95% CI) of new-onset CVD events by COPD status (Objective 1a), also describing the influence of clinical characteristics, previous exacerbations and drug therapy class (Objective 1e).

We will describe CV risk assessments used and calculate the prevalence rate ratio (Poisson/negative binomial regression) of CVD risk score assessments and absolute risk scores by COPD status (Objectives 1b,1c). The influence of clinical characteristics, previous exacerbations and drug therapy class will also be investigated (Objective 1d).

COPD vs comorbid COPD-CVD: Time from exacerbation to maintenance therapy change and incidence of CVD (Aim 2)

From the main analysis file, a sub-population of patients who have COPD and ≥1 exacerbation at follow-up will be further investigated for the second aim of CARDIO-PULSE study. Baseline characteristics of the population will be described for each of the covariates and outcomes. People will enter the study when they have their first

exacerbation during the observation period ('index date') and will exit at 12 months (or at their first event for the time-to-event analyses).

Time from COPD exacerbations to COPD maintenance therapy change (Objectives 2a,2b) by comorbid CVD status will be assessed using time-to-event analyses (Cox regression and Kaplan-Meier plots), also stratified by therapy class at baseline and class regimen. No therapy changes will be treated as censored data at 12 months post-index date. The findings will be presented using graphs and hazard ratios (HR), with 95% confidence intervals (CI).

Impact from time to maintenance therapy change on subsequent occurrence of CVD events by comorbid CVD status (Objective 2c) will be assessed using rate-based analyses (Poisson or negative binomial regression with 95% CI) to produce incidence rate ratios (IRR) for comorbid COPD-CVD compared with COPD alone.

5.2. Sample size

For the sample size calculations, a secondary analysis was conducted using data from preliminary investigation (conference proceedings) on high-risk COPD patients and age-gender-smoking-matched controls using OPCRDA data [26]. The analysis comprised 3,862 individuals with high-risk COPD on 1 Jan 2014. A feasibility count on the OPCRDA data has identified 5,997 patients with COPD on 1 Jan 2020. To allow for missing/data exclusions (e.g., indeterminate gender) and patients who cannot be matched, we have conservatively assumed that there will be 5,500 patients in the final analysis from 1 Jan 2020.

COPD vs no COPD: CVD events and CVD risk score assessments (Aim 1)

In the secondary analysis of OPCRDA data [26], the rate of stroke, heart failure and myocardial infarction in the age-gender-smoking-matched control arm was 0.04 (n=126), 0.05 (n=151), and 0.02 (n=55), respectively. The IRR for COPD vs control patients was 1.17 (0.94,1.46) for stroke; 1.40 (1.17,1.68) for heart failure; and 2.17 (1.60,2.94) for myocardial infarction. Mean follow-up was 3.8 years.

Assuming a similar rate of CVD conditions from 1 Jan 2020, a sample size of 5,500 in each arm gives 92% power to detect an IRR of 1.17, given no overdispersion (i.e., Poisson assumptions met), mean duration of 3.8 years and a 2-tailed 0.05 level of significance. With the same criteria and an expected IRR of 1.40, gives >99% power for heart failure and an IRR of 2.17 gives >99% power for myocardial infarction.

Using prevalence estimates from the team's previous work (conference proceedings) on CVD risk scores [27], 11.7% (rate 0.12; 603/5157) patients with COPD and 2.4% (rate 0.02; 9296/386803) patients without COPD had a CVD risk score, which gives an IRR of 4.90. The same rates and follow-up duration give >99% power to detect a prevalence rate ratio of 4.90.

COPD vs comorbid COPD-CVD: Time from exacerbation to maintenance therapy change and incidence of CVD (Aim 2)

In the secondary analysis of OPCRDA data [26], a total of 1,611 individuals of 3,862 (41.7%) with COPD had an exacerbation (treated as 'index date') within 5 years (from Jan 2014 to Dec 2019). If we assume similar rates from Jan 2020 among 5,500 individuals and a maintenance therapy change rate of 50% within 12 months (to align with GOLD 2026 guidance to consider a treatment change after an exacerbation [9]), this sample size (n=2,293) is sufficient to detect a 22% greater risk (HR: 1.22) in comorbid CVD-COPD patients (compared with COPD alone), assuming a moderate correlation between outcome and covariates (r^2 = 0.3) with a power of 80% and a 5% significance level (2-sided).

5.3. Software

Analyses for this study will be conducted in Stata v15.1 [28].

6. REGULATORY AND ETHICAL COMPLIANCE

Approvals will be sought from the Anonymised Data Ethics and Protocol Transparency (ADEPT) Committee prior to study commencement. The study will be registered on the

European Network of Centres for Pharmacoepidemiology and Pharmacovigilance (ENCePP).

7. DATA DISSEMINATION

As well as the two peer-reviewed publications arising from this work, data will be disseminated widely via the project team and steering committee's extensive networks, including the International Cardiovascular and Respiratory Alliance (ICRA), the Thoracic Society of Australia and New Zealand (TSANZ), The Royal Australian College of General Practitioners (RACGP), Australian College of Rural and Remote Medicine (ACRRM), Primary Health Networks (PHN), Respiratory Effectiveness Group (REG), European Respiratory Society (ERS), American Thoracic Society (ATS) and Primary Care Respiratory Society (PCRS). We will also disseminate findings via social media and Optimum Patient Care Australia (OPCA) press release.

8. TIMELINES

Action	Timeline
Protocol drafted for internal review	Nov 2025
Protocol for AZ review	Dec 2025
Steering committee protocol review and kick-off meeting	Jan 2026
Final draft protocol for AZ	Feb 2026
Final analyses – slide deck	Apr 2026
Study report	Jul 2026
Study manuscript 1	Dec 2026
Study manuscript 2	Dec 2026

9. VERSION HISTORY

Version	Date	Authors
1.1	13 Nov 2025	Freya Tyrer
1.2	18 Nov 2025	Ziggy Burnett-Kirton, Freya Tyrer, John Busby
1.3	20 Nov 2025	Ziggy Burnett-Kirton, Freya Tyrer
2.0	26 Nov 2025	Victoria Carter, John Busby
3.0	4 Dec 2025	Victoria Carter, John Busby, Freya Tyrer
3.1	12 Dec 2025	Angela Catanzariti, Freya Tyrer
4.0	2 Feb 2026	Steering committee members, Freya Tyrer, Ziggy Burnett-Kirton, Alexander Roussos, Porsche le Cheng, John Busby, David Price
4.1	12 Feb 2026	Changes after meeting steering committee, Freya Tyrer, Ziggy Burnett-Kirton, Alexander Roussos, Porsche le Cheng, John Busby, Victoria Carter, David Price

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