

length of 24 min (range: 9–65 min). The main barriers and enablers identified mapped to five domains of the deprescribing framework. Barriers included insufficient resources of time, staff and technology, lack of co-ordination between health-care settings and negative social pressure from LTCF colleagues. Additional barriers exist in private LTCFs including insufficient deprescribing awareness, commitment and the need for incentives. Deprescribing enablers included education, interprofessional support, and involving patients in deprescribing decision making. The importance of 'buy-in' from all stakeholders was emphasised. To encourage deprescribing, potential enablers include HCP education, pharmacist role expansion and tailored deprescribing guidelines within a structured process.

Conclusion: Interventions to support deprescribing should build on existing systems, involve stakeholders and utilise guidelines within a structured process. A deprescribing algorithm, supported with tailored guidelines and education could encourage engagement from all HCPs, both during formal medication reviews and for any change in a patient's clinical condition, requiring deprescribing. Expanding pharmacists' role to include deprescribing responsibilities may help to overcome time constraints for other HCPs. Patient engagement in the form of shared decision making is an option to overcome the anticipated negative response from patients and representatives toward deprescribing. Any intervention must account for the nuanced barriers and enablers which exist in both public and private settings.

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Predicting antipsychotic-induced weight gain in first episode psychosis – a protocol for a field-wide systematic review of non-genetic prognostic factor studies

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Introduction: Obesity prevalence is 2-3-fold higher amongst those with schizophrenia, compared to the general population.¹ Antipsychotic-induced weight gain (AIWG) is undoubtedly a significant contributor to such high obesity rates.^{1,2} Aside from serious physiological consequences, AIWG can negatively impact quality of life,³ and is a common cause of premature antipsychotic discontinuation and future reluctance to engage in treatment.⁴ Managing AIWG is complex and challenging for patients, clinicians and policy makers alike. This is partly due to extensive interindividual variability associated with this side effect – both in susceptibility to initial weight gain and the extent of total weight gained following antipsychotic initiation.⁵ Identification of prognostic factors associated with a subsequent clinical outcome can help distinguish groups of people with a different than average prognosis and thus inform personalised management.⁶ Risk-stratified information highlighting those at increased risk of experiencing significant weight gain prior to antipsychotic treatment would facilitate a move towards more personalised patient care, including individualised weight monitoring and management protocols. Availability of reliable prognostic information may also inform personalised antipsychotic prescribing through avoidance of higher risk antipsychotics amongst those with a worse prognosis.

Aims: This protocol is for a planned systematic review to identify the current quantity, quality and clinical utility of baseline clinical, sociodemographic, and biological prognostic factors in predicting the likelihood of significant AIWG occurring prior to antipsychotic commencement.

Methods: The cohort included will be previously antipsychotic-naïve adults experiencing a first episode of psychosis and where antipsychotic treatment begins concomitantly with prognostic factor measurement and weight monitoring. Studies included will be both randomised and prospective non-randomised studies and will be identified through electronic searching of four databases and two trial registers, followed by reference searching, forward citation searching, and liaison with content experts. A meta-analysis of studies will be conducted if valid data are available assessing associations between a baseline prognostic factor and a pre-specified anthropometric



outcome in three or more studies deemed sufficiently homogeneous. Both unadjusted and adjusted estimates of all prognostic factor associations will be eligible for assessment. A random effects model will be applied given the likely significant heterogeneity of eligible studies. The Grading of Recommendations Assessment, Development and Evaluation (GRADE) framework modified for prognostic factor research will be used to assess evidence certainty.⁷ This protocol was prepared in accordance with the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) Protocols guideline,⁸ and latest guidance from the Prognosis Methods Group of the Cochrane Collaboration.⁹

Conclusion: Potential application of review results include improving treatment recommendations and individual patient management, for example, identifying those more likely to benefit from preventative and early interventions to mitigate AIWG and improve both the quality and experience of care. Further potential applications of identifying reliable prognostic factors include informing development and optimisation of prognostic models to predict individual risk, including combination with reliable genetic prognostic factors, identifying potential predictors of treatment response, and aiding in the design and analysis of intervention studies through stratified randomisation ensuring balanced treatment groups across levels of a prognostic factor.⁶

Registration details: PROSPERO registration number CRD42021258148.

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Effect of inhaled corticosteroid use in smokers and non-smokers with COPD

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Introduction: Inhaled corticosteroids (ICS) have long been a treatment option for Chronic Obstructive Pulmonary Disease (COPD). However, questions over the efficacy of ICS have persisted and recent changes to guidance¹ have stated that further research is needed to predict the factors that indicate ICS responsiveness. One such factor is smoking status.²

Aim: To explore the effect of inhaled corticosteroid use in smokers versus non- and ex-smokers with COPD using primary care data.

Methods: A prospective cohort study using data from the Clinical Practice Research Datalink. Patients with a new diagnosis of COPD between 2006 and 2016 were identified and categorised by ICS usage; 'strict' use ($\geq 80\%$ persistence/year) and 'non-use' ($< 10\%$ persistence/year). Smoking status was recorded at the beginning of the study. Patients were followed for up to 5 years after their index date and matching of ICS users to non-ICS users using propensity score was performed. Propensity score matching was based on sex, age at diagnosis, other COPD medication use, Charlson score, index of multiple deprivation and asthma co-diagnosis.

Results: A cohort of 46,617 people with COPD was identified; 16 560 were 'strict' ICS users, of which 5989 (36.2%) were current smokers, and 30 057 were non-ICS users of which 14,595 (48.6%) were current smokers.

Lung function: At year five, current smokers/ICS users had a 106 ml (95% CI -0.173 to -0.038 , $p = 0.002$) decline in lung function compared to non-ICS users. Ex- and non-smokers/ICS users had a 48 ml (95% CI -0.091 to -0.005 , $p = 0.027$) decline versus non-ICS users.

Exacerbations: At year five, current smokers/ICS users had an increase of 0.091 exacerbations/year (95% CI 0.015 to 0.167; $p = 0.019$) compared to non-ICS users. Ex/never smokers had an increase of 0.075 exacerbations/year (95% CI 0.017 to 0.133; $p = 0.011$) versus non-ICS users.

Conclusion: Smoking with ICS use leads to worse outcomes in terms of lung function and yearly exacerbations than with no ICS use. Ex- and non-smokers using ICS also had worse outcomes than with no ICS use, but this deterioration was less than the smokers; suggesting that using ICS while smoking is of limited benefit in COPD. Limitations of this study are the use of real-world data, of which there were missing entries and potentially lack of rigour when data was initially entered.

