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COPD

1

Int J Chron Obstruct Pulmon Dis

. 2022 Aug 22;17:1909-1920.

doi: 10.2147/COPD.S369804. eCollection 2022.

The Effect of Maintenance Treatment with Erdosteine on Exacerbation Treatment and Health Status in Patients with COPD: A Post-Hoc Analysis of the RESTORE Dataset

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Affiliations expand

- PMID: 36034589

- PMCID: PMC9416404

- DOI: 10.2147/COPD.S369804

Abstract

Purpose: To explore the effect of erdosteine on COPD exacerbations, health-related quality of life (HRQoL), and subjectively assessed COPD severity.

Patients and methods: This post-hoc analysis of the RESTORE study included participants with COPD and spirometrically moderate (GOLD 2; post-bronchodilator forced expiratory volume in 1 second [FEV_1] 50–79% predicted; n = 254), or severe airflow limitation (GOLD 3; post-bronchodilator FEV_1 30–49% predicted; n = 191) who received erdosteine 300 mg twice daily or placebo added to usual maintenance therapy for 12 months. Antibiotic and oral corticosteroid use was determined together with patient-reported HRQoL (St George's Respiratory Questionnaire, SGRQ). Patient and physician subjective COPD severity scores (scale 0–4) were rated at baseline, 6 and 12 months. Data were analyzed using descriptive statistics for exacerbation severity, COPD severity, and treatment group. Comparisons between treatment groups used Student's *t*-tests or ANCOVA as appropriate.

Results: Among GOLD 2 patients, 43 of 126 erdosteine-treated patients exacerbated (7 moderate-to-severe exacerbations), compared to 62 of 128 placebo-treated patients (14 moderate-to-severe exacerbations). Among those with moderate-to-severe exacerbations, erdosteine-treated patients had a shorter mean duration of corticosteroid treatment (11.4 days vs 13.3 days for placebo, $P = 0.043$), and fewer patients required antibiotic treatment with/without oral corticosteroids (71.4% vs 85.8% for placebo, $P < 0.001$). Erdosteine-treated GOLD 2 patients who exacerbated showed significant improvements from baseline in SGRQ total scores and subjective disease severity scores (patient- and physician-rated), compared with placebo-treated patients regardless of exacerbation severity. Among GOLD 3 patients, there were no significant differences between treatment groups on any of these measures.

Conclusion: Adding erdosteine to the usual maintenance therapy of COPD patients with moderate airflow limitation reduced the number of exacerbations, the duration of treatment with corticosteroids and the episodes requiring treatment with antibiotics. Additionally, treatment with erdosteine improved HRQoL and patient-reported disease severity.

Keywords: COPD exacerbation; antibiotic; chronic obstructive pulmonary disease; erdosteine; health-related quality of life; systemic corticosteroid.

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Conflict of interest statement

P Calverley reports personal fees from Recipharm, during the conduct of the study; personal fees from Edmond Pharma, Novartis, Phillips Respironics, and Genentech, outside the submitted work. P Rogliani participated as a lecturer and advisor in scientific meetings sponsored by Almirall, AstraZeneca, Biofutura, Boehringer Ingelheim, Chiesi Farmaceutici, GlaxoSmithKline (GSK), Menarini Group, MSD, Mundipharma, Novartis, Edmond Pharma and Roche. Her department was funded by Almirall, Boehringer Ingelheim, Chiesi Farmaceutici, Novartis, and Zambon. J Wedzicha reports grants from AstraZeneca, personal fees from Chiesi Farmaceutici, Novartis, grants, personal fees from GSK, grants from Boehringer Ingelheim, personal fees from Gilead, grants from Genentech, outside the submitted work. A Papi reports grants from Chiesi Farmaceutici, AstraZeneca, GSK, Boehringer Ingelheim, Teva, Sanofi, personal fees from Chiesi Farmaceutici, AstraZeneca, GSK, Novartis, Sanofi, IQVIA, Avillion, Elpen Pharmaceuticals, MSD, Boehringer Ingelheim, Menarini, Zambon, Mundipharma, Teva, Edmond Pharma, outside the submitted work. M Cazzola and C Page are consultants to Edmond Pharma who manufactures and markets erdosteine. C Page reports personal fees from Edmond Pharma, during the conduct of the study; equity from Verona Pharma, personal fees from Glycos Innovation, personal fees from Eurodrug, personal fees from worldwide clinical trial, outside the submitted work; and Non Executive Director of Epiendo Pharmaceuticals. AF Cicero reported personal fees from Edmond Pharma. The authors report no other conflicts of interest in this work.

- 29 references
- 4 figures

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Respiration

. 2022 Aug 26;1-9.

doi: 10.1159/000525865. Online ahead of print.

Long-Term Noninvasive Ventilation in Chronic Obstructive Pulmonary Disease: Association between Clinical Phenotypes and Survival

Jean-Paul Janssens[1,2], Chloé Cantero[1], Patrick Pasquina[1], Cyril Jaksic[3], Dan Adler[1,2], Christophe Uldry[4], Bernard Egger[4], Maura Prella[5], Alain Bigin Younossian[6], Claudio Rabec[7], Paola M Soccal Gasche[1,2], Jean-Louis Pépin[8,9], all the investigators of the Geneva Lake Study

Affiliations expand

- PMID: 36030774

- DOI: 10.1159/000525865

Abstract

Background: Long-term noninvasive ventilation (LTNIV) is widely used in patients with chronic hypercapnic respiratory failure (CHRF) related to COPD. Prognosis of these patients is however poor and heterogenous.

Research question: In COPD patients under LTNIV for CHRF, is it possible to identify specific phenotypes which are predictive of probability of pursuing NIV and survival?

Study design and methods: A latent class analysis was performed in a COPD population under LTNIV included in a comprehensive database of patients in the Geneva Lake area, to determine clinically relevant phenotypes. The observation period of this subgroup of COPD was extended to allow assessment of survival and/or pursuit of NIV for at least 2 years after inclusion. A logistic regression was conducted to generate an equation accurately attributing an individual patient to a defined phenotype. The identified phenotypes were compared on a series of relevant variables, as well as for probability of pursuing NIV or survival. A competitive risk analysis allowed to distinguish death from other causes of cessation of NIV.

Results: Two phenotypes were identified: a "respiratory COPD" profile with very severe airway obstruction, a low or normal body mass index, and a low prevalence of comorbidities and a "systemic COPD" profile of obese COPDs with moderate airway obstruction and a high rate of cardiovascular and metabolic comorbidities. The logistic regression correctly classified 95.7% of patients studied. Probability of pursuing NIV and survival were significantly related to these phenotypes, with a poorer prognosis for

"respiratory COPD." Probability of death 5 years after implementing NIV was 22.3% (95% CI: 15.4-32.2) for "systemic COPD" versus 47.2% (37.4-59.6) for "respiratory COPD" (p = 0.001).

Conclusion: The two distinct phenotypes of COPD under LTNIV for CHRF identified appear to be strongly related to prognosis and require further validation in other cohort studies.

Keywords: Chronic hypercapnic respiratory failure; Chronic obstructive pulmonary disease; Home mechanical ventilation; Latent class analysis; Long-term mechanical ventilation; Overlap syndrome; Phenotypes; Prognosis; Survival.

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Eur Geriatr Med

. 2022 Aug 27.

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Probable sarcopenia: associations with common geriatric syndromes and comorbidities in Turkish geriatric patients from a university hospital

Duygu Erbas Sacar[1], Cihan Kılıc[2], Meryem Merve Oren[3], Tugba Erdogan[2], Serdar Ozkok[2], Caglar Ozer Aydın[2], Nezahat Muge Catikkas[2], Mehmet Akif Karan[2], Gulistan Bahat[2]

Affiliations expand

- PMID: 36029439

- DOI: 10.1007/s41999-022-00691-9

Abstract

Purpose: EWGSOP2 defines "probable sarcopenia" as the presence of low muscle strength without non-muscle causes. The associations of probable sarcopenia have been studied in few reports to date, and our intention in this study is to identify associations of probable sarcopenia with common geriatric syndromes in a sample of older adults who attended the geriatric outpatient clinic of Istanbul University Hospital.

Methods: The present study was designed as a retrospective cross-sectional study. We performed a comprehensive geriatric assessment to the participants. Univariate analyses were performed to determine relationship of probable sarcopenia with age, sex, common geriatric syndromes, i.e., frailty, falls, polypharmacy, malnutrition, and comorbidities, i.e., diabetes mellitus, hypertension, chronic kidney disease, chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF), depression, osteoporosis, and the variables found to be significant were included in logistic regression analyses. The results are presented as an odds ratio (OR), with a 95% confidence interval (CI).

Results: Included in the study were 456 participants with a mean age of 74.6 ± 6.6 years, of which 71.1% were female. Probable sarcopenia was identified in 12.7% (n = 58) of the sample. A multivariate analysis was carried out, the factors associated with probable sarcopenia were identified as male sex (OR 0.269, 95% CI 0.142-0.510), frailty (OR 4.265, 95% CI 2.200-8.267) and chronic kidney disease (OR 3.084, 95% CI 1.105-8.608).

Conclusion: Probable sarcopenia was more significantly associated with frailty than with other geriatric syndromes, signifying its importance as a marker for frailty. The study further identified chronic renal failure as a factor significantly associated with probable sarcopenia among the variety of studied diseases that frequently accompany aging.

Keywords: Comorbidity; Frailty; Probable sarcopenia; Sarcopenia.

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- 64 references

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Trials

. 2022 Aug 26;23(1):707.

doi: 10.1186/s13063-022-06603-3.

The effects of breathing exercises and inhaler training in patients with COPD on the severity of dyspnea and life quality: a randomized controlled trial

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Affiliations expand

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- PMCID: PMC9419340

- DOI: 10.1186/s13063-022-06603-3

Free PMC article

Abstract

Background: Severe dyspnea and poor quality of life are common in chronic obstructive pulmonary disease (COPD). The most important reason for this is wrong applications in inhaler treatment. In addition, inhaler treatments that support non-pharmacological methods increase the effectiveness of the drug. The aim of this study was to determine the effects of breathing exercises and inhaler training for chronic obstructive pulmonary disease patients on the severity of dyspnea and life quality.

Methods: The research was a randomized controlled trial. A total of 67 patients with COPD were included. The patients were randomized into two groups. Intervention group 1 were given pursed lip breathing exercise and inhaler training and Intervention group 2 were given only inhaler training. A follow-up after 4 weeks was carried out in both groups. Patient outcomes in both groups were assessed by a COPD assessment test (CAT), the Modified Medical Research Council (mMRC) scale, and the St. George's Respiratory

Questionnaire scale (SGRQ). This study followed the CONSORT checklist for randomized controlled trials. In the data analysis, independent t, Mann-Whitney U, ANOVA, Wilcoxon analysis, and Pearson chi-square tests were used.

Results: The pursed lips exercise and inhaler drug use skills of patients in both groups increased ($p<0.001$). The median value of the CAT and mMRC scores were statistically significant for both groups ($p<0.005$). The mean of life quality scores of patients in both groups decreased, and this result was found to be statistically significant in all sub-dimensions and in the total scale score for both groups ($p<0.001$). Although the increase in the quality of life and the decrease in the severity of dyspnea of the patients in both groups were significant, neither group was superior to the other ($p>0.05$).

Conclusions: As a result of the study, it was found that the skill of using the inhaler and the life quality of the patients increased, and the severity of dyspnea decreased. Supporting inhaler treatments with non-pharmacological methods can increase drug efficacy and quality of life.

Trial registration: ClinicalTrials.gov NCT04739488. Registered on 21 Feb 2021.

Keywords: Breathing exercises; COPD; Dyspnea; Inhaler training; Quality of life; Randomized controlled trial.

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Conflict of interest statement

The authors declare no competing interests.

- 34 references
- 1 figure

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Associated data, Grant supportexpand

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Eur Respir J

. 2022 Aug 25;2200469.

doi: 10.1183/13993003.00469-2022. Online ahead of print.

Association of respiratory symptoms and lung function with occupation in the multinational Burden of Obstructive Lung Disease (BOLD) study

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Affiliations expand

- PMID: 36028253

- DOI: 10.1183/13993003.00469-2022

Abstract

Chronic obstructive pulmonary disease has been associated with exposures in the workplace. We aimed to assess the association of respiratory symptoms and lung function with occupation in the Burden of Obstructive Lung Disease study. We analysed cross-sectional data from 28,823 adults (≥40 years) in 34 countries. Eleven occupations were considered and grouped by likelihood of exposure to organic dusts, inorganic dusts and fumes. The association of chronic cough, chronic phlegm, wheeze, dyspnoea, FEV1/FVC and FVC with occupation was assessed, per study site, using multivariable regression. These estimates were then meta-analysed. Sensitivity analyses explored differences

between sexes and gross national income (GNI). Overall, working in settings with potentially high exposure to dusts or fumes was associated with respiratory symptoms but not lung function differences. The most common occupation was farming. Compared to people not working in any of the 11 considered occupations, those who were farmers for ≥20 years were more likely to have chronic cough (OR=1.52, 95%CI 1.19-1.94), wheeze (OR=1.37, 95%CI 1.16-1.63), and dyspnoea (OR=1.83, 95%CI 1.53-2.20), but not lower FVC (β=0.02L, 95%CI -0.02L to 0.06L) or lower FEV_1/FVC (β=0.04%, 95%CI -0.49% to 0.58%). Some findings differed by sex and GNI. In summary, at a population level, the occupational exposures considered in this study do not appear to be major determinants of differences in lung function, although they associate with more respiratory symptoms. As not all work settings were included in this study, respiratory surveillance should still be encouraged among high-risk dusty and fume job workers, especially in low- and middle-income countries.

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Review

J Med Internet Res

. 2022 Aug 26;24(8):e37100.

doi: 10.2196/37100.

Telehealth for the Longitudinal Management of Chronic Conditions: Systematic Review

Allison A Lewinski[1,2], Conor Walsh[1,3], Sharron Rushton[2], Diana Soliman[4], Scott M Carlson[4], Matthew W Luedke[5,6], David J Halpern[3,7], Matthew J Crowley[1,4], Ryan J Shaw[2], Jason A Sharpe[1], Anastasia-Stefania Alexopoulos[1,4], Amir Alishahi Tabriz[8], Jessica R Dietch[9], Diya M Uthappa[10], Soohyun Hwang[11], Katharine A Ball Ricks[12], Sarah Cantrell[13], Andrzej S Kosinski[14], Belinda Ear[1], Adelaide M Gordon[1], Jennifer M Gierisch[1,3,15], John W Williams Jr[1,3,16], Karen M Goldstein[1,3]

Affiliations expand

- PMID: 36018711

- DOI: 10.2196/37100

Free article

Abstract

Background: Extensive literature support telehealth as a supplement or adjunct to in-person care for the management of chronic conditions such as congestive heart failure (CHF) and type 2 diabetes mellitus (T2DM). Evidence is needed to support the use of telehealth as an equivalent and equitable replacement for in-person care and to assess potential adverse effects.

Objective: We conducted a systematic review to address the following question: among adults, what is the effect of synchronous telehealth (real-time response among individuals via phone or phone and video) compared with in-person care (or compared with phone, if synchronous video care) for chronic management of CHF, chronic obstructive pulmonary disease, and T2DM on key disease-specific clinical outcomes and health care use?

Methods: We followed systematic review methodologies and searched two databases (MEDLINE and Embase). We included randomized or quasi-experimental studies that evaluated the effect of synchronously delivered telehealth for relevant chronic conditions that occurred over ≥2 encounters and in which some or all in-person care was supplanted by care delivered via phone or video. We assessed the bias using the Cochrane Effective Practice and Organization of Care risk of bias (ROB) tool and the certainty of evidence using the Grading of Recommendations Assessment, Development, and Evaluation. We described the findings narratively and did not conduct meta-analysis owing to the small number of studies and the conceptual heterogeneity of the identified interventions.

Results: We identified 8662 studies, and 129 (1.49%) were reviewed at the full-text stage. In total, 3.9% (5/129) of the articles were retained for data extraction, all of which (5/5, 100%) were randomized controlled trials. The CHF study (1/5, 20%) was found to have high ROB and randomized patients (n=210) to receive quarterly automated asynchronous web-based review and follow-up of telemetry data versus synchronous personal follow-up (in-person vs phone-based) for 1 year. A 3-way comparison across study arms found no

significant differences in clinical outcomes. Overall, 80% (4/5) of the studies (n=466) evaluated synchronous care for patients with T2DM (ROB was judged to be low for 2, 50% of studies and high for 2, 50% of studies). In total, 20% (1/5) of the studies were adequately powered to assess the difference in glycosylated hemoglobin level between groups; however, no significant difference was found. Intervention design varied greatly from remote monitoring of blood glucose combined with video versus in-person visits to an endocrinology clinic to a brief, 3-week remote intervention to stabilize uncontrolled diabetes. No articles were identified for chronic obstructive pulmonary disease.

Conclusions: This review found few studies with a variety of designs and interventions that used telehealth as a replacement for in-person care. Future research should consider including observational studies and studies on additional highly prevalent chronic diseases.

Keywords: chronic obstructive; delivery of health care; diabetes mellitus, type 2; heart failure; pulmonary disease; systematic review; telemedicine; veterans.

©Allison A Lewinski, Conor Walsh, Sharron Rushton, Diana Soliman, Scott M Carlson, Matthew W Luedke, David J Halpern, Matthew J Crowley, Ryan J Shaw, Jason A Sharpe, Anastasia-Stefania Alexopoulos, Amir Alishahi Tabriz, Jessica R Dietch, Diya M Uthappa, Soohyun Hwang, Katharine A Ball Ricks, Sarah Cantrell, Andrzej S Kosinski, Belinda Ear, Adelaide M Gordon, Jennifer M Gierisch, John W Williams Jr, Karen M Goldstein. Originally published in the Journal of Medical Internet Research (https://www.jmir.org), 26.08.2022.

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J Clin Med

. 2022 Aug 22;11(16):4926.

doi: 10.3390/jcm11164926.

Utility of Initial Arterial Blood Gas in Neuromuscular versus Non-Neuromuscular Acute Respiratory Failure in Intensive Care Unit Patients

Ahmad R Abuzinadah[1,2], Asma Khaled Almalki[3], Rinad Zuwaimel Almuteeri[3], Rahaf Hassan Althalabi[3], Hanin Abdullah Sahli[3], Fatima Abdulrahman Hayash[3], Rahaf Hamed Alrayiqi[3], Seraj Makkawi[4,5,6], Alaa Maglan[4], Loujen O Alamoudi[4], Noof M Alamri[7], Maha H Alsaati[3], Aysha A Alshareef[1,2], Sultan Saeed Aljereish[8], Ahmed K Bamaga[9], Faris Alhejaili[10], Ahmad Abdulaziz Abulaban[7,11,12], Mohammed H Alanazy[8]

Affiliations expand

- PMID: 36013163

- PMCID: PMC9410118

- DOI: 10.3390/jcm11164926

Free PMC article

Abstract

Background: The arterial blood gas (ABG) parameters of patients admitted to intensive care units (ICUs) with acute neuromuscular respiratory failure (NMRF) and non-NMRF have not been defined or compared in the literature. **Methods:** We retrospectively collected the initial ABG parameters (pH, $PaCO_2$, PaO_2, and HCO_3) of patients admitted to ICUs with acute respiratory failure. We compared ABG parameter ranges and the prevalence of abnormalities in NMRF versus non-NMRF and its categories, including primary pulmonary disease (PPD) (chronic obstructive pulmonary disease, asthma, and bronchiectasis), pneumonia, and pulmonary edema. **Results:** We included 287 patients (NMRF, n = 69; non-NMRF, n = 218). The difference between NMRF and non-NMRF included the median (interquartile range (IQR)) of pH (7.39 (7.32-7.43), 7.33 (7.22-7.39), p < 0.001), PaO_2 (86.9 (71.4-123), 79.6 (64.6-99.1) mmHg, p = 0.02), and HCO_3 (24.85 (22.9-27.8), 23.4 (19.4-26.8) mmol/L, p = 0.006). We found differences in the median of $PaCO_2$ in NMRF (41.5 mmHg) versus PPD (63.3 mmHg), PaO_2 in NMRF (86.9 mmHg) versus pneumonia (74.3 mmHg), and

HCO_3 in NMRF (24.8 mmol/L) versus pulmonary edema (20.9 mmol/L) (all *p* < 0.01). NMRF compared to non-NMRF patients had a lower frequency of hypercarbia (24.6% versus 39.9%) and hypoxia (33.8% versus 50.5%) (all *p* < 0.05). NMRF compared to PPD patients had lower frequency of combined hypoxia and hypercarbia (13.2% versus 37.8%) but more frequently isolated high bicarbonate (33.8% versus 8.9%) (all *p* < 0.001). **Conclusions:** The ranges of ABG changes in NMRF patients differed from those of non-NMRF patients, with a greater reduction in PaO_2 in non-NMRF than in NMRF patients. Combined hypoxemia and hypercarbia were most frequent in PPD patients, whereas isolated high bicarbonate was most frequent in NMRF patients.

Keywords: Guillain–Barré syndrome; amyotrophic lateral sclerosis; blood gas; myasthenia gravis; neuromuscular; pulmonary; respiratory failure.

Conflict of interest statement

The authors declare no conflict of interest.

- 21 references
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Review

Respir Res

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. 2022 Aug 23;23(1):213.

doi: 10.1186/s12931-022-02123-5.

Prognostic risk factors for moderate-to-severe exacerbations in patients with chronic obstructive pulmonary disease: a systematic literature review

John R Hurst[1], MeiLan K Han[2], Barinder Singh[3], Sakshi Sharma[4], Gagandeep Kaur[3], Enrico de Nigris[5], Ulf Holmgren[6], Mohd Kashif Siddiqui[3]

Affiliations expand

- PMID: 35999538

- PMCID: PMC9396841

- DOI: 10.1186/s12931-022-02123-5

Free PMC article

Abstract

Background: Chronic obstructive pulmonary disease (COPD) is a leading cause of morbidity and mortality worldwide. COPD exacerbations are associated with a worsening of lung function, increased disease burden, and mortality, and, therefore, preventing their occurrence is an important goal of COPD management. This review was conducted to identify the evidence base regarding risk factors and predictors of moderate-to-severe exacerbations in patients with COPD.

Methods: A literature review was performed in Embase, MEDLINE, MEDLINE In-Process, and the Cochrane Central Register of Controlled Trials (CENTRAL). Searches were conducted from January 2015 to July 2019. Eligible publications were peer-reviewed journal articles, published in English, that reported risk factors or predictors for the occurrence of moderate-to-severe exacerbations in adults age ≥ 40 years with a diagnosis of COPD.

Results: The literature review identified 5112 references, of which 113 publications (reporting results for 76 studies) met the eligibility criteria and were included in the review. Among the 76 studies included, 61 were observational and 15 were randomized controlled

clinical trials. Exacerbation history was the strongest predictor of future exacerbations, with 34 studies reporting a significant association between history of exacerbations and risk of future moderate or severe exacerbations. Other significant risk factors identified in multiple studies included disease severity or bronchodilator reversibility (39 studies), comorbidities (34 studies), higher symptom burden (17 studies), and higher blood eosinophil count (16 studies).

Conclusions: This systematic literature review identified several demographic and clinical characteristics that predict the future risk of COPD exacerbations. Prior exacerbation history was confirmed as the most important predictor of future exacerbations. These prognostic factors may help clinicians identify patients at high risk of exacerbations, which are a major driver of the global burden of COPD, including morbidity and mortality.

Keywords: Biomarkers; COPD; Comorbidities; Exacerbations; Hospitalization; Predictors; Systematic literature review.

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Conflict of interest statement

JRH reports consulting fees from AstraZeneca; speaker fees from AstraZeneca, Chiesi, Pfizer, and Takeda; and travel support from GlaxoSmithKline and AstraZeneca. MKH reports assistance with conduction of this research and publication from AstraZeneca; personal fees from Aerogen, Altesa Biopharma, AstraZeneca, Boehringer Ingelheim, Chiesi, Cipla, DevPro, GlaxoSmithKline, Integrity, Medscape, Merck, Mylan, NACE, Novartis, Polarean, Pulmonx, Regeneron, Sanofi, Teva, Verona, United Therapeutics, and UpToDate; either in kind research support or funds paid to the institution from the American Lung Association, AstraZeneca, Biodesix, Boehringer Ingelheim, the COPD Foundation, Gala Therapeutics, the NIH, Novartis, Nuvaira, Sanofi, and Sunovion; participation in Data Safety Monitoring Boards for Novartis and Medtronic with funds paid to the institution; and stock options from Altesa Biopharma and Meissa Vaccines. BS, GK, and MKS are former employees of Parexel International. SS is an employee of Parexel International, which was funded by AstraZeneca to conduct this analysis. EdN is a former employee of AstraZeneca and previously held stock and/or stock options in the company. UH is an employee of AstraZeneca and holds stock and/or stock options in the company.

- 111 references
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Meta-Analysis

Respiration

. 2022 Aug 23;1-11.

doi: 10.1159/000525980. Online ahead of print.

Inhaled Corticosteroids and Mycobacterial Infection in Patients with Chronic Airway Diseases: A Systematic Review and Meta-Analysis

Yajie You[1], Yingmeng Ni[2], Guochao Shi[2,3]

Affiliations expand

- PMID: 35998604

- DOI: 10.1159/000525980

Abstract

Background: Inhaled corticosteroids (ICSs) have been widely used in chronic airway diseases, such as asthma, chronic obstructive pulmonary disease (COPD), and bronchiectasis. However, whether ICS use causes mycobacterial infection is uncertain. Some conclusions of published studies were inconsistent.

Objective: We aimed to investigate the association between the use of ICSs and mycobacterial infections in patients with chronic airway diseases.

Methods: This review was registered on PROSPERO (CRD42021284607). We focused on examining the association between ICS use and mycobacterial infection (nontuberculous mycobacterial [NTM] infection as well as tuberculosis [TB]). We searched PubMed (MEDLINE), Sciencenet, Cochrane, and EMBASE databases for studies up to 2021 to retrieve articles. The enrollment conditions included gender, enrollment diagnosis and ICS use in chronic airway disease patients, and so on. Preclinical studies, review articles, editorials, reviews, conference abstracts, and book chapters were excluded. Methodologically, the study was assessed using the Newcastle Ottawa Scale, and Rev-man5 was used for statistical analysis.

Results: Ten studies (including 4 NTM and 6 TB articles) with 517,556 patients met the inclusion criteria and were included in this meta-analysis. From the NTM pooled analyses, ICS use was associated with increased odds of NTM infection in patients with chronic airway diseases (odds ratio [OR] = 3.93, 95% confidence interval [CI] 2.12-7.27), subgroup analysis showed that high-dose ICS use (OR = 2.27, 95% CI 2.08-2.48) and fluticasone use (OR = 2.42, 95% CI 2.23-2.63) were associated with increased odds of NTM infection risk in patients with chronic respiratory diseases. The TB pooled analyses showed a significant association between ICS use and risk of TB infection in patients with chronic respiratory diseases (OR = 2.01, 95% CI 1.23-3.29). Subgroup analysis showed that in chronic respiratory diseases, ICS use increased odds of TB infection in high-dose ICS use (OR = 1.70, 95% CI 1.56-1.86) and in COPD patients (OR = 1.45, 95% CI 1.29-1.63).

Conclusion: Our meta-analysis indicated that ICS use may increase the odds of mycobacterial infection in chronic respiratory disease patients, and this conclusion is more applicable to patients with high dose of ICS or fluticasone in NTM infection subgroups. In addition, high-dose ICS use may have higher risk of TB infection in patients with chronic respiratory diseases, especially COPD. Therefore, we should be vigilant about the application of ICS use in chronic respiratory diseases to avoid infection.

Keywords: Asthma; Bronchiectasis; Chronic obstructive pulmonary disease; Chronic respiratory diseases; Inhaled corticosteroids; Mycobacterium.

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J Innate Immun

. 2022 Aug 23;1-16.

doi: 10.1159/000526080. Online ahead of print.

Expansion of Phenotypically Altered Dendritic Cell Populations in the Small Airways and Alveolar Parenchyma in Patients with Chronic Obstructive Pulmonary Disease

Michiko Mori[1], Carl-Magnus Clausson[1], Caroline Sanden[2], Jimmie Jönsson[2], Cecilia K Andersson[1], Premkumar Siddhuraj[1], Medya Shikhagaie[1], Karolina Åkesson[1], Anders Bergqvist[3], Claes-Göran Löfdahl[3], Jonas S Erjefält[1,3]

Affiliations expand

- PMID: 35998572

- DOI: 10.1159/000526080

Free article

Abstract

Contrasting the antigen-presenting dendritic cells (DCs) in the conducting airways, the alveolar DC populations in human lungs have remained poorly investigated. Consequently, little is known about how alveolar DCs are altered in diseases such as chronic obstructive pulmonary disease (COPD). This study maps multiple tissue DC categories in the distal lung

across COPD severities. Specifically, single-multiplex immunohistochemistry was applied to quantify langerin/CD207+, CD1a+, BDCA2+, and CD11c+ subsets in distal lung compartments from patients with COPD (GOLD stage I-IV) and never-smoking and smoking controls. In the alveolar parenchyma, increased numbers of CD1a+langerin- ($p < 0.05$) and BDCA-2+ DCs ($p < 0.001$) were observed in advanced COPD compared with controls. Alveolar CD11c+ DCs also increased in advanced COPD ($p < 0.01$). In small airways, langerin+ and BDCA-2+ DCs were also significantly increased. Contrasting the small airway DCs, most alveolar DC subsets frequently extended luminal protrusions. Importantly, alveolar and small airway langerin+ DCs in COPD lungs displayed site-specific marker profiles. Further, multiplex immunohistochemistry with single-cell quantification was used to specifically profile langerin DCs and reveal site-specific expression patterns of the maturation and activation markers S100, fascin, MHC2, and B7. Taken together, our results show that clinically advanced COPD is associated with increased levels of multiple alveolar DC populations exhibiting features of both adaptive and innate immunity phenotypes. This expansion is likely to contribute to the distal lung immunopathology in COPD patients.

Keywords: Antigen presentation; Immune pathology; Peripheral lung.

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Respir Res

. 2022 Aug 22;23(1):212.

doi: 10.1186/s12931-022-02144-0.

Prevalence, characteristics, and risk of exacerbation in young patients with chronic obstructive pulmonary disease

Yong Suk Jo[1], Kyung Joo Kim[1], Chin Kook Rhee[1], Kwang Ha Yoo[2], Ki-Suck Jung[3], Yong-Bum Park[4]

Affiliations expand

- PMID: 35996171
- PMCID: PMC9396900
- DOI: 10.1186/s12931-022-02144-0

Free PMC article

Abstract

Background and objective: Early identification of chronic obstructive pulmonary disease (COPD) in young individuals could be beneficial to attempt preventive interventions. The objective of this study was to investigate clinical features and outcomes of young individuals with COPD from the general population cohort.

Methods: We included individuals from the Korean National Health and Nutrition Examination Survey (KNHANES) with spirometry and identifiable smoking status. Young subjects with COPD were defined as aged between 40 and 50 years and had baseline forced expiratory volume in 1 s [FEV_1]/forced vital capacity [FVC] ratio less than 0.7. Outcomes include the risk of exacerbation and medical expenses during 3 years of follow-up.

Results: Among 2236 individuals aged between 40 and 50 years, 95 (4.2%) had COPD, including 36 who were never-smokers and 59 who were ever-smokers. Approximately 98% of COPD subjects had mild to moderate airflow limitation. Inhaler treatment was given to only 6.3% patients in the COPD group. The risk of exacerbation for a 3-year period was analyzed using the never-smoker, non-COPD group as a comparator. Hazards ratio for exacerbation was 1.60 (95% confidence interval [CI] 0.18-14.20) in the never-smoker COPD group and 1.94 (95% CI 0.31-12.07) in the ever-smoker COPD group of young subjects. COPD related medical costs were not significantly different between non-COPD and COPD groups of young individuals.

Conclusions: The risk of exacerbation showed an increasing trend in COPD patients regardless of smoking status compared to non-COPD. More attention to early identification and provision of preventive measures are needed to reduce disease progression and improve outcome.

Keywords: Exacerbation; Medical cost; Smoking; Young patients with COPD.

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Conflict of interest statement

The authors declare that they have no competing interests.

- 36 references
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Nat Microbiol

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. 2022 Sep;7(9):1361-1375.

doi: 10.1038/s41564-022-01196-8. Epub 2022 Aug 22.

Multi-omics analyses of airway host-microbe interactions in chronic

obstructive pulmonary disease identify potential therapeutic interventions

Zhengzheng Yan[#1,2], Boxuan Chen[#1], Yuqiong Yang[#3], Xinzhu Yi[#1], Mingyuan Wei[#1], Gertrude Ecklu-Mensah[4], Mary M Buschmann[4], Haiyue Liu[5], Jingyuan Gao[1], Weijie Liang[1], Xiaomin Liu[1], Junhao Yang[1], Wei Ma[6], Zhenyu Liang[3], Fengyan Wang[3], Dandan Chen[7], Lingwei Wang[7], Weijuan Shi[3], Martin R Stampfli[8], Pan Li[9], Shenhai Gong[10], Xia Chen[11], Wensheng Shu[1], Emad M El-Omar[9], Jack A Gilbert[4], Martin J Blaser[12], Hongwei Zhou[13], Rongchang Chen[14], Zhang Wang[15]

Affiliations expand

- PMID: 35995842

- DOI: 10.1038/s41564-022-01196-8

Abstract

The mechanistic role of the airway microbiome in chronic obstructive pulmonary disease (COPD) remains largely unexplored. We present a landscape of airway microbe-host interactions in COPD through an in-depth profiling of the sputum metagenome, metabolome, host transcriptome and proteome from 99 patients with COPD and 36 healthy individuals in China. Multi-omics data were integrated using sequential mediation analysis, to assess in silico associations of the microbiome with two primary COPD inflammatory endotypes, neutrophilic or eosinophilic inflammation, mediated through microbial metabolic interaction with host gene expression. Hypotheses of microbiome-metabolite-host interaction were identified by leveraging microbial genetic information and established metabolite-human gene pairs. A prominent hypothesis for neutrophil-predominant COPD was altered tryptophan metabolism in airway lactobacilli associated with reduced indole-3-acetic acid (IAA), which was in turn linked to perturbed host interleukin-22 signalling and epithelial cell apoptosis pathways. In vivo and in vitro studies showed that airway microbiome-derived IAA mitigates neutrophilic inflammation, apoptosis, emphysema and lung function decline, via macrophage-epithelial cell cross-talk mediated by interleukin-22. Intranasal inoculation of two airway lactobacilli restored IAA and recapitulated its protective effects in mice. These findings provide the rationale for therapeutically targeting microbe-host interaction in COPD.

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- 97 references

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Review

Am J Physiol Cell Physiol

. 2022 Aug 22.

doi: 10.1152/ajpcell.00292.2022. Online ahead of print.

Impaired regenerative capacity contributes to skeletal muscle dysfunction in chronic obstructive pulmonary disease (COPD)

Ariel Jaitovich[1]

Affiliations expand

- PMID: 35993519

- DOI: 10.1152/ajpcell.00292.2022

Abstract

Locomotor skeletal muscle dysfunction is a relevant comorbidity of chronic obstructive pulmonary disease (COPD) and is strongly associated with worse clinical outcomes including higher mortality. Over the last decades, a large body of literature helped characterize the process, defining the disruptive muscle phenotype caused by COPD which involves reduction in muscle mass, force-generation capacity, fatigue-tolerance, and regenerative potential following injury. A major limitation in the field has been the scarcity of well-calibrated animal models to conduct mechanistic research based on loss- and gain-of-function studies. This article provides an overall description of the process, the tools available to mechanistically investigate it, and the potential role of mitochondrially-driven metabolic signals on the regulation muscle regeneration after injury in COPD. Finally, a description of future avenues to further expand on the area is proposed based on very recent evidence involving mitochondrial metabolic cues affecting myogenesis.

Keywords: autophagy; chronic pulmonary diseases; myogenesis; pulmonary emphysema; satellite cells.

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Endocr Connect

. 2022 Aug 22;11(9):e220289.

doi: 10.1530/EC-22-0289. Print 2022 Sep 1.

Effects and safety of metformin in patients with concurrent diabetes mellitus and chronic obstructive

pulmonary disease: a systematic review and meta-analysis

Ziting Liang[1,2], Mengge Yang[1,2], Changjuan Xu[1,2], Rong Zeng[1,2], Liang Dong[1,2]

Affiliations expand

- PMID: 35900801

- DOI: 10.1530/EC-22-0289

Free article

Abstract

Aim: This study aimed to investigate the effects and safety of metformin in patients with concurrent diabetes mellitus (DM) and chronic obstructive pulmonary disease (COPD).

Methods: PubMed, Embase, Web of Science, the China National Knowledge, and Cochrane Database were searched to find studies that examined the effects and safety of metformin in patients with concurrent DM and COPD. We conducted a meta-analysis with a risk ratio (RR) and assessed the quality of included studies and pooled evidence.

Results: Eight studies were involved. Metformin was associated with lower risk of COPD-related hospitalizations (RR: 0.72, 95% CI: 0.53-0.98; I2= 89%) and all-cause mortality (RR: 0.60, 95% CI: 0.36-1.01, I2= 69%) in patients with concurrent DM and COPD, but did not increase the risk of hyperlactatemia (RR: 1.14, 95% CI: 0.92-1.41, I2 = 8%).

Conclusions: Metformin use is associated with lower risk of COPD-related hospitalizations and risk of all-cause mortality without increasing the risk of hyperlactatemia. Considerations should be given to conduct more high-quality randomized trials involving larger samples.

Keywords: chronic obstructive pulmonary disease; diabetes mellitus; meta-analysis; metformin; safety.

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1

Curr Med Res Opin

. 2022 Aug 27;1-13.

doi: 10.1080/03007995.2022.2100649. Online ahead of print.

Over-prescription of short-acting β $_2$-agonists is associated with poor asthma outcomes: results from the African cohort of the SABINA III study

Adel Khattab[1], Ashraf Madkour[1], Anish Ambaram[2], Clifford Smith[3], Chakaya J Muhwa[4,5], Jared O Mecha[6], Mohamed Alsayed[7], Maarten J H I Beekman[8]

Affiliations expand

- PMID: 36031882

- DOI: 10.1080/03007995.2022.2100649

Abstract

Background: The extent of short-acting β_2-agonist (SABA) overuse in Africa remains poorly documented. As part of the SABA use IN Asthma (SABINA) III study, we assessed SABA prescriptions/clinical outcomes in 3 African countries.

Methods: Data on disease characteristics/asthma treatments were collected from patients (≥12 years) using electronic case report forms. Patients were classified by investigator-defined asthma severity (guided by the 2017 Global Initiative for Asthma) and practice type (primary/specialist care). Multivariable regression models analyzed associations between SABA prescriptions and outcomes.

Results: Data from 1778 patients (mean age, 43.7 years) were analyzed. Most patients were female (62.4%) and had moderate-to-severe asthma (63.3%), with 57.1 and 42.9% of patients treated in specialist and primary care, respectively. Asthma was partly controlled/uncontrolled in 66.2% of patients, with 57.9% experiencing ≥1 severe exacerbation in the previous 12 months. Overall, 46.5% of patients were prescribed ≥3 SABA canisters in the preceding 12 months (over-prescription); 26.2% were prescribed ≥10 canisters. SABAs were purchased over-the-counter by 32.6% of patients, of whom 79.3% had received SABA prescriptions; 71.9% and 40.1% for ≥3 and ≥10 canisters, respectively. Higher SABA prescriptions (vs. 1-2 canisters) were associated with increased incidence rate of severe exacerbations and lower odds of having at least partly controlled asthma (except 3-5 canisters).

Conclusions: Findings from this African cohort of the SABINA III study indicate that SABA over-prescription and SABA over-the-counter purchase are common and associated with poor asthma-related outcomes. This highlights the need for healthcare providers/policymakers to align clinical practices with the latest treatment recommendations.

Keywords: Asthma; asthma control; burden; exacerbation; prescription; short-acting β2-agonist.

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Int Immunopharmacol

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. 2022 Aug 25;112:109180.

doi: 10.1016/j.intimp.2022.109180. Online ahead of print.

Inhaled delivery of recombinant interferon-lambda restores allergic inflammation after development of asthma by controlling Th2- and Th17-cell-mediated immune responses

Jina Won[1], Ara Jo[1], Chan Hee Gil[1], Sujin Kim[1], Haeun Shin[1], Hyun Jik Kim[2]

Affiliations expand

- PMID: 36030690

- DOI: 10.1016/j.intimp.2022.109180

Abstract

Remarkable progress has recently been achieved to identify the biological function and potential value of novel therapeutic targets for the effective control of allergic asthma. Interferon (IFN)-λ has been suggested to restrict chronic inflammation in the lungs of asthmatic mice and we sought to determine the contribution of IFN-λ as an asthma therapeutic. We show that inhaled IFN-λ can restrict Th2 and Th17 inflammation in the lungs of asthmatic mice, accompanied with alteration of IL-10 secretion. BALB/C mice were used for an asthmatic mouse model with OVA. Recombinant IFN-λs (IFN-λ_2: 2 μg, IFN-λ_3: 2 μg) were inoculated into asthmatic mice after OVA challenge by intranasal delivery. Lungs of asthmatic mice were severely inflamed, with extensive inflammatory cell infiltration and increased goblet cell metaplasia with higher total lung resistance. Transcription of IL-4, IL-5, IL-13, and IL-17A was significantly higher until five days after the final OVA challenge. Asthmatic mice were administered recombinant IFN-λ via inhalation three times after the last challenge and the asthmatic mice showed improvement in lung histopathologic findings, and total lung resistance was maintained under normal range. IFN-λ inhalation exhibited significant decreases in Th2 and Th17 cytokine levels, and the populations of Th2 and Th17 cells were recovered from the lungs of asthmatic mice. Additionally, increase in IL-10 secretion from CD4 + Th cells population was observed in response to inhaled delivery of IFN-λ along with alterations in Th2 and Th17 cell-derived inflammation. Our findings show that inhaled delivery of IFN-λ can restrict airway inflammation in the lungs of asthmatic mice by controlling Th2- and Th17-mediated responses accompanied by regulation of IL-10 secretion even after asthma development.

Keywords: Asthma; IFN-λ; IL-10; Inhaled delivery; Th17; Th2.

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Am J Respir Crit Care Med

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. 2022 Aug 27.

doi: 10.1164/rccm.202207-1419LE. Online ahead of print.

Mechanisms of Obesity-related Asthma: Is Insulin Getting on Your Nerves?

Zhenying Nie[1], Allison D Fryer[2], David B Jacoby[3], Matthew G Drake[4]

Affiliations expand

- PMID: 36029301

- DOI: 10.1164/rccm.202207-1419LE

No abstract available

Keywords: hyperinsulinemia; insulin resistance; obesity-related asthma.

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Am J Respir Crit Care Med

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. 2022 Aug 27.

doi: 10.1164/rccm.202208-1585LE. Online ahead of print.

Reply to: Mechanisms of Obesity-related Asthma: Is Insulin Getting on Your Nerves?

Michael C Peters[1], Mark Schiebler[2], David T Mauger[3], John V Fahy[4]

Affiliations expand

- PMID: 36029296

- DOI: 10.1164/rccm.202208-1585LE

No abstract available

Keywords: asthma; gluocse; insulin; metabolic; obesity.

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J Allergy Clin Immunol Pract

. 2022 Aug 23;S2213-2198(22)00651-1.

doi: 10.1016/j.jaip.2022.06.036. Online ahead of print.

Dupilumab Is Effective in Patients With Moderate-to-Severe Uncontrolled GINA-Defined Type 2 Asthma Irrespective of an Allergic Asthma Phenotype

Klaus F Rabe[1], J Mark FitzGerald[2], Eric D Bateman[3], Mario Castro[4], Ian D Pavord[5], Jorge F Maspero[6], William W Busse[7], Kenji Izuhara[8], Nadia Daizadeh[9], Benjamin Ortiz[10], Nami Pandit-Abid[11], Paul J Rowe[11], Yamo Deniz[10]

Affiliations expand

- PMID: 36028446

- DOI: 10.1016/j.jaip.2022.06.036

Abstract

Background: The Global Initiative for Asthma report recommends consideration of add-on biologics for patients with type 2 inflammation (blood eosinophils ≥150 cells/μL, fractional exhaled nitric oxide [Feno] ≥20 parts per billion or allergic asthma) whose asthma cannot be controlled by high-dose inhaled corticosteroids. In QUEST (NCT02414854), add-on dupilumab versus placebo was efficacious in patients with uncontrolled, moderate to severe asthma, including those with eosinophils greater than or equal to 150 cells/μL and/or Feno greater than or equal to 25 parts per billion.

Objective: To assess dupilumab efficacy in patients with a type 2 phenotype in the presence or absence of allergic asthma phenotype.

Methods: Patients aged 12 years or older received add-on dupilumab 200/300 mg versus matched placebo every 2 weeks for 52 weeks. Allergic asthma phenotype was defined as baseline serum total IgE greater than or equal to 30 IU/mL and 1 or more perennial aeroallergen-specific IgE level greater than or equal to 0.35 kU/L. Annualized rate of severe asthma exacerbations and changes from study baseline in prebronchodilator and postbronchodilator FEV_1 were evaluated in patients with allergic and nonallergic phenotype with baseline blood eosinophils greater than or equal to 150 cells/μL and/or Feno greater than or equal to 20 parts per billion.

Results: Of 1902 patients in QUEST, 83.3% had eosinophils and/or Feno above Global Initiative for Asthma thresholds; 56.9% had evidence for allergic asthma. Dupilumab significantly reduced the rate of severe asthma exacerbations in patients with (48.8%) and without (64.0%) evidence of allergic asthma and improved prebronchodilator and postbronchodilator FEV_1 in patients with elevated type 2 biomarkers, irrespective of whether they showed evidence of an allergic asthma phenotype.

Conclusions: In patients with type 2 biomarkers over Global Initiative for Asthma thresholds, dupilumab significantly reduced exacerbations and improved lung function. Efficacy was not impacted by allergic status.

Keywords: Allergic asthma phenotype; Dupilumab; Exacerbations; Lung function; Type 2 inflammation.

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Case Reports

Clin Case Rep

. 2022 Aug 22;10(8):e6255.

doi: 10.1002/ccr3.6255. eCollection 2022 Aug.

Omalizumab for the treatment of severe allergic asthma in children: A tale of two

Sinéad Brannick[1], Mary McDonald[1], Peter Greally[1], Basil Elnazir[1], Oneza Ahmareen[1]

Affiliations expand

- PMID: 36017116

- PMCID: PMC9393874

- DOI: 10.1002/ccr3.6255

Free PMC article

Abstract

Omalizumab is a monoclonal antibody which targets immunoglobulin E. It is approved as an add-on therapy for children with severe allergic asthma. Assessment of endotype and phenotype is necessary in order to correctly identify those patients who are most likely to respond to omalizumab. Children with severe asthma represent a complex heterogeneous group. This report outlines the background, management, and outcomes for two children initiated on omalizumab for severe allergic asthma in Children's Health Ireland at Tallaght. It demonstrates the difficulties faced by this cohort and the positive impact targeted biological therapy can have. Given the substantial cohort of children with asthma attending our tertiary center, it also indicates that comprehensive stepwise care can achieve adequate control in the vast majority of cases without the requirement for additional therapies.

Keywords: allergy and immunology; asthma; biologic therapies; pediatrics and adolescent medicine; respiratory medicine.

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Conflict of interest statement

There are no conflicts of interest to declare.

- 18 references

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J Clin Med

. 2022 Aug 22;11(16):4926.

doi: 10.3390/jcm11164926.

Utility of Initial Arterial Blood Gas in Neuromuscular versus Non-Neuromuscular Acute Respiratory Failure in Intensive Care Unit Patients

Ahmad R Abuzinadah[1,2], Asma Khaled Almalki[3], Rinad Zuwaimel Almuteeri[3], Rahaf Hassan Althalabi[3], Hanin Abdullah Sahli[3], Fatima Abdulrahman Hayash[3], Rahaf Hamed Alrayiqi[3], Seraj Makkawi[4,5,6], Alaa Maglan[4], Loujen O Alamoudi[4], Noof M Alamri[7], Maha H Alsaati[3], Aysha A Alshareef[1,2], Sultan Saeed Aljereish[8], Ahmed K Bamaga[9], Faris Alhejaili[10], Ahmad Abdulaziz Abulaban[7,11,12], Mohammed H Alanazy[8]

Affiliations expand

- PMID: 36013163

- PMCID: PMC9410118

- DOI: 10.3390/jcm11164926

Free PMC article

Abstract

Background: The arterial blood gas (ABG) parameters of patients admitted to intensive care units (ICUs) with acute neuromuscular respiratory failure (NMRF) and non-NMRF have not been defined or compared in the literature. **Methods:** We retrospectively collected the initial ABG parameters (pH, $PaCO_2$, PaO_2, and HCO_3) of patients admitted to ICUs with acute respiratory failure. We compared ABG parameter ranges and the prevalence of abnormalities in NMRF versus non-NMRF and its categories, including primary pulmonary disease (PPD) (chronic obstructive pulmonary disease, asthma, and bronchiectasis), pneumonia, and pulmonary edema. **Results:** We included 287 patients (NMRF, $n = 69$; non-NMRF, $n = 218$). The difference between NMRF and non-NMRF included the median (interquartile range (IQR)) of pH (7.39 (7.32-7.43), 7.33 (7.22-7.39), p < 0.001), PaO_2 (86.9 (71.4-123), 79.6 (64.6-99.1) mmHg, $p = 0.02$), and HCO_3 (24.85 (22.9-27.8), 23.4 (19.4-26.8) mmol/L, $p = 0.006$). We found differences in the median of $PaCO_2$ in NMRF (41.5 mmHg) versus PPD (63.3 mmHg), PaO_2 in NMRF (86.9 mmHg) versus pneumonia (74.3 mmHg), and HCO_3 in NMRF (24.8 mmol/L) versus pulmonary edema (20.9 mmol/L) (all p < 0.01). NMRF compared to non-NMRF patients had a lower frequency of hypercarbia

(24.6% versus 39.9%) and hypoxia (33.8% versus 50.5%) (all *p* < 0.05). NMRF compared to PPD patients had lower frequency of combined hypoxia and hypercarbia (13.2% versus 37.8%) but more frequently isolated high bicarbonate (33.8% versus 8.9%) (all *p* < 0.001). **Conclusions:** The ranges of ABG changes in NMRF patients differed from those of non-NMRF patients, with a greater reduction in PaO_2 in non-NMRF than in NMRF patients. Combined hypoxemia and hypercarbia were most frequent in PPD patients, whereas isolated high bicarbonate was most frequent in NMRF patients.

Keywords: Guillain–Barré syndrome; amyotrophic lateral sclerosis; blood gas; myasthenia gravis; neuromuscular; pulmonary; respiratory failure.

Conflict of interest statement

The authors declare no conflict of interest.

- 21 references
- 2 figures

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Allergy Asthma Clin Immunol

. 2022 Aug 25;18(1):78.

doi: 10.1186/s13223-022-00719-6.

Inhaled corticosteroids do not affect the antibody titer against the SARS-

CoV-2 spike protein in BNT162b2 mRNA vaccinated patients

Takeo Nakajima[1], Tatsuya Nagano[2], Yoshiharu Miyata[3], Shoko Murakami[4], Satoshi Mitsuyuki[5], Yohei Funakoshi[6], Kimikazu Yakushijin[6], Hitoshi Horimoto[7], Yoshihiro Nishimura[8], Kazuyuki Kobayashi[4]

Affiliations expand

- PMID: 36008820
- PMCID: PMC9403962
- DOI: 10.1186/s13223-022-00719-6

Free PMC article

Abstract

Objectives: Oral corticosteroids reduce the antibody titer of the BNT162b2 mRNA vaccine against SARS-CoV-2. To date, the effect of inhaled corticosteroids on antibody titers is unknown.

Study design: The design of this study is retrospective study.

Methods: We analyzed the relationship between the clinical features and total antibody titers against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) spike protein in 320 subjects who had never been infected with Coronavirus disease 2019 (COVID-19) and were vaccinated the second time with the BNT162b2 mRNA vaccine between October 1 to December 28, 2021.

Results: Of the 320 subjects, 205 were treated with inhaled corticosteroids. The median antibody titer of patients treated with inhaled corticosteroids was 572 U/mL, which was significantly higher than that of patients treated without inhaled corticosteroids (454U/mL, P = 0.00258). The median antibody titers of smokers, men, and patients aged 65 years and over, were 315.5 U/mL, 385 U/mL, and 425.5 U/mL, respectively. These results are significantly lower than those of patients who never smoked, women, and patients aged less than 64 years (582 U/mL [P < 0.0001], 682.5 U/mL [P < 0.0001], and 717 U/mL [P < 0.0001], respectively). The multivariate analysis revealed that females and age were independent antibody titer-reducing factors (P = 0.0001 and P < 0.0001, respectively).

Conclusions: The use of inhaled corticosteroids did not reduce the antibody titer against SARS-CoV-2 spike protein. Clinicians should continue treatment with inhaled corticosteroids if indicated.

Keywords: Asthma; BNT162b2 mRNA vaccine; Inhaled corticosteroid; SARS-CoV-2.

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Conflict of interest statement

The authors declare no competing interests about the study.

- 21 references
- 4 figures

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Nat Med

. 2022 Aug 25.

doi: 10.1038/d41591-022-00088-y. Online ahead of print.

Asthma treatment for those who need it most

Karen O'Leary

- PMID: 36008685

- DOI: 10.1038/d41591-022-00088-y

No abstract available

Keywords: Asthma; Clinical trials; Paediatrics.

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Review

Eur J Clin Pharmacol

. 2022 Aug 26.

doi: 10.1007/s00228-022-03374-3. Online ahead of print.

Use of ketamine in patients with refractory severe asthma exacerbations: systematic review of prospective studies

Luigi La Via[#1,2], Filippo Sanfilippo[#3], Giuseppe Cuttone[4], Veronica Dezio[3,4], Monica Falcone[5], Serena Brancati[6], Claudia Crimi[7], Marinella Astuto[3]

Affiliations expand

- PMID: 36008492

- DOI: 10.1007/s00228-022-03374-3

Abstract

Purpose: Asthma is a heterogeneous disease with a wide range of symptoms. Severe asthma exacerbations (SAEs) are characterized by worsening symptoms and bronchospasm requiring emergency department visits. In addition to conventional strategies for SAEs (inhaled β-agonists,

anticholinergics, and systemic corticosteroids), another pharmacological option is represented by ketamine. We performed a systematic review to explore the role of ketamine in refractory SAEs.

Methods: We performed a systematic search on PubMed and EMBASE up to August 12th, 2021. We selected prospective studies only, and outcomes of interest were oxygenation/respiratory parameters, clinical status, need for invasive ventilation and effects on weaning.

Results: We included a total of seven studies, five being randomized controlled trials (RCTs, population range 44-92 patients). The two small prospective studies (n = 10 and n = 11) did not have a control group. Four studies focused on adults, and three enrolled a pediatric population. We found a large heterogeneity regarding sample size, age and gender distribution, inclusion criteria (different severity scores, if any) and ketamine dosing (bolus and/or continuous infusion). Of the five RCTs, three compared ketamine to placebo, while one used fentanyl and the other aminophylline. The outcomes evaluated by the included studies were highly variable. Despite paucity of data and large heterogeneity, an overview of the included studies suggests absence of clear benefit produced by ketamine in patients with refractory SAE, and some signals towards side effects.

Conclusion: Our systematic review does not support the use of ketamine in refractory SAE. A limited number of prospective studies with large heterogeneity was found. Well-designed multicenter RCTs are desirable.

Keywords: Aminophylline; Asthma; Bronchospasm; Fentanyl; Inflammation; Mechanical ventilation.

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- 36 references

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Review

. 2022 Aug 25;edpract-2022-324308.

doi: 10.1136/archdischild-2022-324308. Online ahead of print.

How to interpret spirometry in a child with suspected asthma

Anthony Brown[1], Benjamin McNaughten[2], Catherine Russell[3], Patricia Watters[4], Dara O'Donoghue[2,5]

Affiliations expand

- PMID: 36008112

- DOI: 10.1136/archdischild-2022-324308

Abstract

Asthma is one of the most common chronic disorders of childhood. The typical symptoms are a result of reversible airway obstruction. There is no 'gold-standard' test to diagnose asthma, but the most commonly used investigation to help with a diagnosis is spirometry. This article outlines some of the technical aspects of spirometry together with how the forced expiration manoeuver and bronchodilator responsiveness testing can be performed and interpreted in a child with suspected asthma.

Keywords: Adolescent Health; Child Health; Paediatrics; Respiratory Medicine.

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Conflict of interest statement

Competing interests: None declared.

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Allergy Asthma Clin Immunol

. 2022 Aug 24;18(1):76.

doi: 10.1186/s13223-022-00716-9.

The value of clinical observation: sleuthing for allergies on the front lines

Elissa M Abrams[1], Allan Becker[2]

Affiliations expand

- PMID: 36002904

- PMCID: PMC9404553

- DOI: 10.1186/s13223-022-00716-9

Free PMC article

No abstract available

Conflict of interest statement

The authors declare no competing interests.

- 7 references

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Observational Study

BMJ Open

. 2022 Aug 24;12(8):e060160.

doi: 10.1136/bmjopen-2021-060160.

Real-life impact of uncontrolled severe asthma on mortality and healthcare use in adolescents and adults: findings from the retrospective, observational RESONANCE study in France

Nicolas Roche[1], Gilles Garcia[2], Alexandre de Larrard[3], Charlotte Cancalon[3], Stève Bénard[3], Vincent Perez[4], Aymeric Mahieu[4], Laurine Vieu[4], Pascal Demoly[5]

Affiliations expand

- PMID: 36002203

- DOI: 10.1136/bmjopen-2021-060160

Free article

Abstract

Objective: To characterise uncontrolled severe asthma and compare the disease burden with the general and asthmatic populations.

Design: Retrospective observational study using a national sample of a French healthcare database (Echantillon Généraliste des Bénéficiaires (EGB)).

Setting: The EGB, an anonymised permanent sample of health insurance databases, representing 1/97th of the French population.

Participants: Patients (≥12 years) were selected in year 2014 and followed 2 years. A cohort of patients with uncontrolled severe asthma was defined using an algorithm based on peer-reviewed literature and Global Initiative for Asthma recommendations. Index date was the occurrence of the first marker of uncontrolled asthma. This cohort was matched with two control cohorts, general population and asthmatic controls, on baseline characteristics.

Main outcomes measures: Mortality, healthcare use and associated costs were studied in the 2 years of follow-up.

Results: Among 467 716 individuals in the EGB, 16 588 patients with asthma were identified, including 739 (4.5%) with uncontrolled severe disease. The survival probability at 2 years for patients with uncontrolled severe asthma (92.0%) was lower than in the general population cohort (96.6%; relative risk of death: 2.35; 95% CI: 1.70 to 3.29; p<0.0001) and tended to be lower than in the control asthmatic cohort (94.3%; p=0.07). Emergency department visits and hospitalisations were higher in patients with uncontrolled severe asthma than in the general population (64.7% vs 34.9%; p<0.0001) and asthmatic controls (64.7% vs 55.2%; p=0.0002). Other components of healthcare use (medical and paramedical visits, medications) were increased in patients with uncontrolled severe asthma compared with control populations. These increases translated into higher costs (p<0.0001 for both comparisons).

Conclusions: This study demonstrates the huge burden of uncontrolled severe asthma in terms of mortality, morbidity and healthcare resource consumption compared with other patients with asthma and with the general population and emphasises the importance of appropriate management in this high-risk population.

Keywords: Asthma; Epidemiology; HEALTH ECONOMICS.

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Conflict of interest statement

Competing interests: PD has received honoraria and/or research grants from ALK, Mylan-Viatris, Stallergenes, ThermoFisher Scientific, AstraZeneca, GSK, Novartis, Menarini and Regeneron. GG has received honoraria from ALK, Novartis, AstraZeneca, GSK, Sanofi and Chiesi for conferences or advisory board meetings. NR has received research funding from Boehringer Ingelheim, GSK, Pfizer and Novartis and honoraria from Boehringer Ingelheim, Pfizer, Novartis, Teva, GSK, AstraZeneca, Chiesi, Sanofi and Zambon. LV, AM, VP are Sanofi employee and may hold shares and/or stock options in the company. No other disclosures were reported.

SUPPLEMENTARY INFO

Publication types, MeSH termsexpand

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14

Exp Lung Res

. 2022 Aug 24;1-12.

doi: 10.1080/01902148.2022.2115166. Online ahead of print.

Aerosol inhalation of *Mycobacterium vaccae* ameliorates airway structural remodeling in chronic asthma mouse model

Qian-Nan Zhang[1], Huan Xiao[1], Li-Ting Fang[1], Qi-Xiang Sun[2], Lao-Dong Li[2], Si-Yue Xu[2], Chao-Qian Li[1]

Affiliations expand

- PMID: 36001552

- DOI: 10.1080/01902148.2022.2115166

Abstract

Background: Airway remodeling is accepted to be a determining component within the natural history of asthma. Nebulized inhalation of *Mycobacterium vaccae* (*M. vaccae*) has a protective effect on asthmatic mice. However, little is known regarding the effect of *M. vaccae* on airway structural remodeling in asthmatic mice. The purpose of this study was to explore the effect and the underlying mechanism of *M. vaccae* aerosol inhalation on airway structural remodeling in an asthma mouse model. **Methods:** Chronic asthma mouse models were established by ovalbumin

induction. The number of inflammatory cells in bronchoalveolar lavage fluid (BALF), pathological alterations in lung tissue, and levels of associated cytokines (IL-5, IL-13, TNF-α, and ovalbumin-specific immunoglobulin E [OVA-sIgE]) were all assessed after *M. vaccae* therapy. The relative expression of interleukin (IL)-1β, tumor necrosis factor-alpha (TNF-α), nuclear factor kappa B (NF-κB), and Wnt1-induced signaling protein 1 (WISP1) mRNA were detected. Western blotting and immunohistochemistry detected the expression of Wnt/β-catenin pathway-related proteins in lung tissue. **Results:** *M. vaccae* aerosol inhalation relieved airway inflammation, airway hyper-responsiveness, and airway remodeling. *M. vaccae* reduced the levels of IL-5, IL-13, TNF-α, and OVA-sIgE in and downregulated the expression of IL-1β, TNF-α, NF-κB, and WISP1 mRNA in the pulmonary. In addition, *M. vaccae* inhibited the expression of β-catenin, WISP1, and Wnt1 protein and upregulated the expression of glycogen synthase kinase-3beta (GSK-3β). **Conclusion:** Nebulized inhalation of *M. vaccae* can reduce airway remodeling during asthma.

Keywords: Mycobacterium vaccae; Airway remodelling; chronic asthma; inflammation.

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J Investig Allergol Clin Immunol

. 2022 Aug 24;0.

doi: 10.18176/jiaci.0850. Online ahead of print.

Bronchodilator reversibility in the GAN severe asthma cohort

K Milger[1,2], D Skowasch[3], E Hamelmann[4], C Mümmler[1,2], M Idzko[5], M Gappa[6], M Jandl[7], C Körner-Rettberg[8], R Ehmann[9], O Schmidt[10], C Taube[11], A Holtdirk[12], H Timmermann[13], R Buhl[14], S Korn[15,16]

Affiliations expand

- PMID: 36000830

- DOI: 10.18176/jiaci.0850

Abstract

Background and objective: Positive bronchodilator reversibility (BDR) is a diagnostic criterion for asthma. However, patients with asthma may exhibit negative BDR test.To describe frequency of positive and negative BDR in patients with severe asthma and associations with phenotypic characteristics.

Methods: Positive BDR was defined as FEV1 increase > 200 ml AND > 12% upon testing with a short-acting beta-agonist (SABA).

Results: Out of 2013 patients included in the German Asthma Net (GAN) severe asthma registry, 793 had data on BDR. Hereof, 250 (31.5%) had a positive and 543 (68.5%) had a negative BDR test. Comorbidities significantly associated with negative BDR were gastro-esophageal reflux (GERD) (28.0% vs 40.0%, p<0.01) and EGPA (0.4% vs 3.0%; p<0.05), while smoking history (active: 2.8% vs 2.2%; ex: 40.0% vs 41.7%) and COPD comorbidity (5.2% vs 7.2%) were similar in both groups. Patients with positive BDR had worse asthma control (median ACQ-5 3.4 vs 3.0, p<0.05), reported dyspnea at rest (26.8% vs 16.4%, p<0.001) and chest tightness (36.4% vs 26.2%, p<0.001) more frequently, had more severe airway obstruction at baseline (FEV1% pred: 56 vs 64, p<0.001) and higher FeNO levels (41 vs 33 ppb, p<0.05), while diffusion capacity did not differ (DLCO-SB % pred. 70% vs 71%). Multivariate linear regression analysis identified association of lower baseline FEV1% (p<0.001) and chest tightness (p<0.05) with positive, and GERD (p<0.05) with negative BDR.

Conclusion: In this real-life setting the majority of patients with severe asthma exhibited negative BDR. Interestingly, this was not associated with smoking history or COPD, but with lower FeNO and presence of GERD.

Keywords: Bronchodilator responsiveness; FeNO; GERD; Real-life cohort; Severe asthma.

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Review

J Investig Allergol Clin Immunol

. 2022 Aug 24;0.

doi: 10.18176/jiaci.0853. Online ahead of print.

Eosinophilic esophagitis due to aeroallergens: A Systematic Review, update, and our experience

A R Gratacós Gómez[1], E Gómez Torrijos[1]

Affiliations expand

- PMID: 36000828

- DOI: 10.18176/jiaci.0853

Abstract

Eosinophilic esophagitis is a chronic antigen-mediated esophageal disease characterized by symptoms related to esophageal dysfunction and histologically by Th2 inflammation (at least15 eosinophils/high power field) when other secondary systemic and local causes of esophageal eosinophilia are excluded. Though this disease was initially ascribed to a delayed reaction to food allergens, emerging evidence suggests that aeroallergens may also play roles in his pathogenesis and evolution. Some studies support seasonal variation in Eosinophilic esophagitis diagnosis and disease exacerbations about the increase in aeroallergens to which patients are sensitized. It is also known that this disease can be generated after an extensive, identifiable aeroallergen exposure and after treatment with specific immunotherapy with food or aeroallergens. Recently, it has been postulated that treatment of allergic rhinoconjunctivitis may improve Eosinophilic esophagitis symptoms, though data is limited to case reports and small series. Currently, biomarkers and biologic therapies are not helpful for diagnosis or inducing clinical and histological remission of the disease. Although there are high hopes for dupilumab This review aims to give visibility to the involvement of aeroallergens in the triggering and exacerbation of eosinophilic esophagitis since many of them, in addition to being in the air and being able to inhale them, can also ingest them as part of the food. It is essential to highlight that we must try to discover the cause of the disease since it is crucial for its remission.

Keywords: Aeroallergens; Asthma; Eosinophilic esophagitis; Pollen; Rhinitis.

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J Investig Allergol Clin Immunol

. 2022 Aug 24;0.

doi: 10.18176/jiaci.0848. Online ahead of print.

Understanding severe asthma through small and Big Data in Spanish hospitals – PAGE Study

C Melero Moreno[1,2], C Almonacid Sánchez[3], D Bañas Conejero[4], S Quirce[5], F J Álvarez Gutiérrez[6], V Cardona[7], M G Sánchez-Herrero[4], J B Soriano[8], PAGE Study group

Affiliations expand

- PMID: 36000822

- DOI: 10.18176/jiaci.0848

Abstract

Background and objective: Data on severe asthma prevalence is limited. The implementation of Electronic Health records (EHRs) offers a unique research opportunity to test machine (ML) tools in epidemiological studies. The aim was to estimate severe asthma (SA) prevalence amongst the asthmatic patients seen in hospital asthma units, using both ML and traditional research methodologies. Secondary objectives were to describe non-severe asthma (NSA) and SA patients during a follow-up period of 12 months.

Methods: The PAGE study is a multicenter, controlled, observational study conducted in 36 Spanish hospitals and split into two phases: a first cross-sectional phase for the estimation of SA prevalence, and a second, prospective phase (3 visits in 12 months) for the follow-up and characterisation of SA and NSA patients. A sub-study with ML was included in 6 hospitals. This ML tool uses EHRead technology, which extracts clinical concepts from EHRs and standardizes them to SNOMED CT.

Results: : A SA prevalence of 20.1% was obtained amongst asthma patients in Spanish hospitals, compared with 9.7% prevalence by the ML tool. The proportion of SA phenotypes and the features of followed-up patients were consistent with previous studies. The clinical predictions of patients' clinical course was unreliable, while the ML only found two predictive models with discriminatory potential to predict outcomes.

Conclusion: This study is the first to estimate SA prevalence, in a hospital population of asthma patients, and to predict patient outcomes using both standard and ML techniques. These findings offer relevant insights for further epidemiological and clinical research in SA.

Keywords: Severe asthma. Prevalence. Big data. Machine learning. Natural language processing. Predictive models.

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Meta-Analysis

Respir Res

. 2022 Aug 23;23(1):214.

doi: 10.1186/s12931-022-02132-4.

The respiratory microbiota alpha-diversity in chronic lung diseases: first systematic review and meta-analysis

Marta Avalos-Fernandez[1,2], Thibaud Alin[#3,4], Clémence Métayer[#3,4], Rodolphe Thiébaut[3,4,5], Raphaël Enaud[6,7], Laurence Delhaes[6,7,8]

Affiliations expand

- PMID: 35999634

- PMCID: PMC9396807

- DOI: 10.1186/s12931-022-02132-4

Free PMC article

Abstract

Background: While there seems to be a consensus that a decrease in gut microbiome diversity is related to a decline in health status, the associations between respiratory microbiome diversity and chronic lung disease remain a matter of debate. We provide a systematic review and meta-analysis of studies examining lung microbiota alpha-diversity in patients with asthma, chronic obstructive pulmonary disease (COPD), cystic fibrosis (CF) or bronchiectasis (NCFB), in which a control group based on disease status or healthy subjects is provided for comparison.

Results: We reviewed 351 articles on title and abstract, of which 27 met our inclusion criteria for systematic review. Data from 24 of these studies were used in the meta-analysis. We observed a trend that CF patients have a less diverse respiratory microbiota than healthy individuals. However, substantial heterogeneity was present and detailed using random-effects models, which limits the comparison between studies.

Conclusions: Knowledge on respiratory microbiota is under construction, and for the moment, it seems that alpha-diversity measurements are not enough documented to fully understand the link between microbiota and health, excepted in CF context which represents the most studied chronic respiratory disease with consistent published data to link alpha-diversity and lung function. Whether differences in respiratory microbiota profiles have an impact on chronic respiratory disease symptoms and/or evolution deserves further exploration.

Keywords: Alpha-diversity; Asthma; Chronic obstructive respiratory disease; Chronic respiratory diseases; Cystic fibrosis; Factor Analysis of Mixed Data; Human lung bacteriome; Human lung microbiome; Meta-analysis; Non-cystic fibrosis bronchiectasis; Random-effects models.

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Conflict of interest statement

The authors declare that they have no competing interests.

- 62 references
- 3 figures

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BMC Pediatr

. 2022 Aug 23;22(1):499.

doi: 10.1186/s12887-022-03571-0.

Interaction between serum cotinine and body mass index on asthma in the children: a cross-sectional study

Li He[1], Xiaojing Xi[2]

Affiliations expand

- PMID: 35999590

- PMCID: PMC9400283

- DOI: 10.1186/s12887-022-03571-0

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Abstract

Background: The purpose of this study was to explore the interaction between serum cotinine (a marker of environmental tobacco smoke exposure) and body mass index (BMI) on asthma in children.

Methods: This cross-sectional study relied on representative samples of American children included in the National Health and Nutrition Examination Survey in 1999-2018. Multivariate logistic regression analyses were to evaluate the association between serum cotinine level, BMI z-score and asthma. Serum cotinine was dichotomized at 0.0436 ng/mL. Interactions were examined by the estimated joint effect of BMI and serum cotinine levels. We also performed interaction analyses in age and ethnicity subgroups.

Results: Among the 11,504 children aged 3 to 12 years included in the analysis, 15.86% (n = 1852) had childhood asthma, 15.68% (n = 1837) were overweight, and 17.31% (n = 2258) were obese. Compared to low serum cotinine, high serum cotinine was significantly associated with asthma [odds ratio (OR) = 1.190, 95% confidence interval (CI): 1.004-1.410]. Overweight (OR = 1.275,

95%CI: 1.079-1.506) and obesity (OR = 1.636, 95%CI: 1.354-1.977) were significantly associated with asthma compared with normal weight. The adjusted attributable proportion of interaction = 0.206 (95%CI: 0.075-0.337) and the adjusted synergy index = 1.617 (95%CI: 1.126-2.098) indicated that there was a significant synergistic effect of serum cotinine levels and BMI on asthma. In males, females, non-Hispanic White and other Hispanic, there were synergistic interactions between serum cotinine levels and BMI on asthma.

Conclusion: A synergistic interaction between serum cotinine and overweight/obesity on childhood asthma was found. For children with asthma, both intensive weight interventions in overweight or obese children and intensive passive smoking interventions in children exposed to the environment may be important.

Keywords: Asthma; BMI z-score; Children; Serum cotinine.

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Conflict of interest statement

The authors declare that they have no competing interests.

- 30 references
- 2 figures

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Meta-Analysis

Respiration

. 2022 Aug 23;1-11.

doi: 10.1159/000525980. Online ahead of print.

Inhaled Corticosteroids and Mycobacterial Infection in Patients with Chronic Airway Diseases: A Systematic Review and Meta-Analysis

Yajie You[1], Yingmeng Ni[2], Guochao Shi[2,3]

Affiliations expand

- PMID: 35998604

- DOI: 10.1159/000525980

Abstract

Background: Inhaled corticosteroids (ICSs) have been widely used in chronic airway diseases, such as asthma, chronic obstructive pulmonary disease (COPD), and bronchiectasis. However, whether ICS use causes mycobacterial infection is uncertain. Some conclusions of published studies were inconsistent.

Objective: We aimed to investigate the association between the use of ICSs and mycobacterial infections in patients with chronic airway diseases.

Methods: This review was registered on PROSPERO (CRD42021284607). We focused on examining the association between ICS use and mycobacterial infection (nontuberculous mycobacterial [NTM] infection as well as tuberculosis [TB]). We searched PubMed (MEDLINE), Sciencenet, Cochrane, and EMBASE databases for studies up to 2021 to retrieve articles. The enrollment conditions included gender, enrollment diagnosis and ICS use in chronic airway disease patients, and so on. Preclinical studies, review articles, editorials, reviews, conference abstracts, and book chapters were excluded. Methodologically, the study was assessed using the Newcastle Ottawa Scale, and Rev-man5 was used for statistical analysis.

Results: Ten studies (including 4 NTM and 6 TB articles) with 517,556 patients met the inclusion criteria and were included in this meta-analysis. From the NTM pooled analyses, ICS use was associated with increased odds of NTM infection in patients with chronic airway diseases (odds ratio [OR] = 3.93, 95% confidence interval [CI] 2.12-7.27), subgroup analysis showed that high-dose ICS use (OR = 2.27, 95% CI 2.08-2.48) and fluticasone use (OR = 2.42, 95% CI 2.23-2.63) were associated with increased odds of NTM infection risk in patients with chronic respiratory diseases. The TB pooled analyses showed a significant association between ICS use and risk of TB infection in patients with chronic respiratory diseases (OR = 2.01, 95% CI 1.23-3.29). Subgroup analysis showed that in chronic respiratory diseases, ICS use increased odds of TB infection in high-dose ICS use (OR = 1.70, 95% CI 1.56-1.86) and in COPD patients (OR = 1.45, 95% CI 1.29-1.63).

Conclusion: Our meta-analysis indicated that ICS use may increase the odds of mycobacterial infection in chronic respiratory disease patients, and this conclusion is more applicable to patients with high dose of ICS or fluticasone in NTM infection subgroups. In addition, high-dose ICS use may have higher risk of TB infection in patients with chronic respiratory diseases, especially COPD. Therefore, we should be vigilant about the application of ICS use in chronic respiratory diseases to avoid infection.

Keywords: Asthma; Bronchiectasis; Chronic obstructive pulmonary disease; Chronic respiratory diseases; Inhaled corticosteroids; Mycobacterium.

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21

Am J Respir Crit Care Med

. 2022 Aug 23.

doi: 10.1164/rccm.202202-0294OC. Online ahead of print.

U.S. Health Care Spending on Respiratory Diseases, 1996–2016

Kevin I Duan[1], Maxwell Birger[2], David H Au[3], Laura J Spece[4], Laura C Feemster[5,6], Joseph L Dieleman[7]

Affiliations expand

- PMID: 35997678

- DOI: 10.1164/rccm.202202-0294OC

Abstract

Rationale: Respiratory conditions account for a large proportion of health care spending in the United States (US). A full characterization of spending across multiple conditions and over time has not been performed.

Objective: To estimate US health care spending for 11 respiratory conditions from 1996-2016, providing detailed trends and an evaluation of factors associated with spending growth.

Methods: We extracted data from the Institute of Health Metrics and Evaluation's Disease Expenditure Project Database, producing annual estimates in spending for 38 age and sex groups, 7 types of care, and 3 payer types. We performed a decomposition analysis to estimate the change in spending that is associated with changes in each of five factors (population growth, population aging, disease prevalence, service utilization, and service price and intensity).

Measurements and main results: Total spending across all respiratory conditions in 2016 was $170.8 billion (95% CI $164.2-$179.2 billion), increasing by $71.7 billion (95% CI $63.2-$80.8 billion) from 1996. The respiratory conditions with the highest spending in 2016 were asthma and chronic obstructive pulmonary disease (COPD), contributing $35.5 billion (95% CI $32.4-$38.2 billion) and $34.3 billion (95% CI $31.5-$37.3 billion), respectively. Increasing service price and intensity were associated with 81.4% (95% CI 70.3-93.0%) growth from 1996 to 2016.

Conclusions: US spending on respiratory conditions is high, particularly for chronic conditions like asthma and COPD. Our findings suggest that service price and intensity, particularly for pharmaceuticals, should be a key focus of attention for policy makers seeking to reduce health care spending growth.

Keywords: health economics; health expenditures; health policy.

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Am J Physiol Lung Cell Mol Physiol

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-

. 2022 Aug 23.

doi: 10.1152/ajplung.00137.2022. Online ahead of print.

Differential airway remodelling changes were observed in patients with asthma COPD overlap (ACO) compared to asthma and COPD patients alone

Surajit Dey[1], Wenying Lu[1], Heinrich C Weber[1,2], Sally Young[1,3], Josie Larby[1,4], Collin Chia[1,4], Greg Haug[1,4], Samuel James Brake[1], Steve Myers[1], Archana Vijay Gaikwad[1], Prem Bhattarai[1], Prabuddha S Pathinayake[5], Peter A B Wark[5,6], Mathew Suji Eapen[1], Sukhwinder Singh Sohal[1]

Affiliations expand

- PMID: 35997281

- DOI: 10.1152/ajplung.00137.2022

Abstract

Background: Management of ACO patients is clinically challenging due to insufficient evidence of pathological changes in these patients.

Methods: In this cross-sectional study, we evaluated airway remodelling in endobronchial biopsies from total 90 subjects, which included 12 ACO, 14 asthmatics, 12 COPD ex-smokers (ES) and 11 current smokers (CS), 28 healthy controls (HC), and 13 normal lung function smokers (NLFS). Tissue was stained with Masson's Trichrome. Epithelium, goblet cells, reticular basement membrane (RBM), cellularity, lamina propria (LP) and smooth muscle (SM) changes were measured using Image-Pro Plus v7 software.

Results: Differential airway remodelling pattern was seen in ACO patients. A limited change was noted in the ACO epithelium than in other pathological groups. RBM was substantially thicker in ACO patients than in HC ($p<0.0002$) and tended to be thicker than in asthmatics and NLFS. The total RBM cells were higher in ACO than the HC ($p<0.0001$), COPD-CS ($p=0.0559$), -ES ($p=0.0345$), and NLFS ($p<0.0002$), but did not differ from asthmatics. Goblet cells were higher in the ACO than the HC ($p=0.0028$) COPD-ES ($p=0.0081$). The total LP cells in ACO appeared to be higher than HC, COPD-CS, and NLFS but appeared to be lower than asthma. Finally, SM area was significantly lower in the ACO than in asthmatics ($p=0.001$), COPD-CS (=0.0290), and NLFS ($p=0.0011$).

Conclusions: This first comprehensive study suggests that ACO patients had distinguishable tissue remodelling that appeared to be more severe than asthmatics and COPD patients. This study will help in informed decision-making for better patient management in clinical practice.

Keywords: ACO; Asthma; COPD; Smoking; airway remodelling.

SUPPLEMENTARY INFO

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Review

Cochrane Database Syst Rev

. 2022 Aug 22;8(8):CD013485.

doi: 10.1002/14651858.CD013485.pub2.

Pulmonary rehabilitation versus usual care for adults with asthma

Christian R Osadnik[1,2], Ciara Gleeson[3], Vanessa M McDonald[4,5,6], Anne E Holland[7,8,9]

Affiliations expand

- PMID: 35993916

- PMCID: PMC9394585 (available on 2023-08-22)

- DOI: 10.1002/14651858.CD013485.pub2

Abstract

Background: Asthma is a respiratory disease characterised by variable airflow limitation and the presence of respiratory symptoms including wheeze, chest tightness, cough and/or dyspnoea. Exercise training is beneficial for people with asthma; however, the response to conventional models of pulmonary rehabilitation is less clear.

Objectives: To evaluate, in adults with asthma, the effectiveness of pulmonary rehabilitation compared to usual care on exercise performance, asthma control, and quality of life (co-primary outcomes), incidence of severe asthma exacerbations/hospitalisations, mental health, muscle strength, physical activity levels, inflammatory biomarkers, and adverse events.

Search methods: We identified studies from the Cochrane Airways Trials Register, Cochrane Central Register of Controlled Trials (CENTRAL), MEDLINE, Embase, ClinicalTrials.gov, and the World Health Organization International Clinical Trials Registry Platform, from their inception to May 2021, as well as the reference lists of all primary studies and review articles.

Selection criteria: We included randomised controlled trials in which pulmonary rehabilitation was compared to usual care in adults with asthma. Pulmonary rehabilitation must have included a minimum of four weeks (or eight sessions) aerobic training and education or self-management. Co-interventions were permitted; however, exercise training alone was not. DATA COLLECTION AND ANALYSIS: Following the use of Cochrane's Screen4Me workflow, two review authors independently screened and selected trials for inclusion, extracted study characteristics and outcome data, and assessed risk of bias using the Cochrane risk of bias tool. We contacted study authors to retrieve missing data. We calculated between-group effects via mean differences (MD) or standardised mean differences (SMD) using a random-effects model. We evaluated the certainty of evidence using GRADE methodology.

Main results: We included 10 studies involving 894 participants (range 24 to 412 participants (n = 2 studies involving n > 100, one contributing to meta-analysis), mean age range 27 to 54 years). We identified one ongoing study and three studies awaiting classification. One study was synthesised narratively, and another involved participants specifically with asthma-COPD overlap. Most programmes were outpatient-based, lasting from three to four weeks (inpatient) or eight to 12 weeks (outpatient). Education or self-management components included breathing retraining and relaxation, nutritional advice and psychological counselling. One programme was specifically tailored for people with severe asthma. Pulmonary rehabilitation compared to usual care may increase maximal oxygen uptake (VO_2 max) after programme completion, but the evidence is very uncertain for data derived using mL/kg/min (MD between groups of 3.63 mL/kg/min, 95% confidence interval (CI) 1.48 to 5.77; 3 studies; n = 129) and uncertain for data derived from % predicted VO_2 max (MD 14.88%, 95% CI 9.66 to 20.1%; 2 studies; n = 60). The evidence is very uncertain about the effects of pulmonary rehabilitation compared to usual care on incremental shuttle walk test distance (MD between groups 74.0 metres, 95% CI 26.4 to 121.4; 1 study; n = 30). Pulmonary rehabilitation may have little to no effect on VO_2 max at longer-term follow up (9 to 12 months), but the evidence is very uncertain (MD -0.69 mL/kg/min, 95% CI -4.79 to 3.42; I^2 = 49%; 3 studies; n = 66). Pulmonary rehabilitation likely improves functional exercise capacity as measured by 6-minute walk distance, with MD between groups after programme completion of 79.8 metres (95% CI 66.5 to 93.1; 5 studies; n = 529; moderate certainty evidence). This magnitude of mean change exceeds the minimally clinically important difference (MCID) threshold for people with chronic respiratory disease. The evidence is very uncertain about the longer-term effects one year after pulmonary rehabilitation for this outcome (MD 52.29 metres, 95% CI 0.7 to 103.88; 2 studies; n = 42). Pulmonary rehabilitation may result in a small improvement in asthma control compared to usual care as measured by Asthma Control Questionnaire (ACQ), with an MD between groups of -0.46 (95% CI -0.76 to -0.17; 2 studies; n = 93; low certainty evidence); however, data

derived from the Asthma Control Test were very uncertain (MD between groups 3.34, 95% CI -2.32 to 9.01; 2 studies; n = 442). The ACQ finding approximates the MCID of 0.5 points. Pulmonary rehabilitation results in little to no difference in asthma control as measured by ACQ at nine to 12 months follow-up (MD 0.09, 95% CI -0.35 to 0.53; 2 studies; n = 48; low certainty evidence). Pulmonary rehabilitation likely results in a large improvement in quality of life as assessed by the St George's Respiratory Questionnaire (SGRQ) total score (MD -18.51, 95% CI -20.77 to -16.25; 2 studies; n = 440; moderate certainty evidence), with this magnitude of change exceeding the MCID. However, pulmonary rehabilitation may have little to no effect on Asthma Quality of Life Questionnaire (AQLQ) total scores, with the evidence being very uncertain (MD 0.87, 95% CI -0.13 to 1.86; 2 studies; n = 442). Longer-term follow-up data suggested improvements in quality of life may occur as measured by SGRQ (MD -13.4, 95% CI -15.93 to -10.88; 2 studies; n = 430) but not AQLQ (MD 0.58, 95% CI -0.23 to 1.38; 2 studies; n = 435); however, the evidence is very uncertain. One study reported no difference between groups in the proportion of participants who experienced an asthma exacerbation during the intervention period. Data from one study suggest adverse events attributable to the intervention are rare. Overall risk of bias was most commonly impacted by performance bias attributed to a lack of participant blinding to knowledge of the intervention. This is inherently challenging to overcome in rehabilitation studies. AUTHORS' CONCLUSIONS: Moderate certainty evidence shows that pulmonary rehabilitation is probably associated with clinically meaningful improvements in functional exercise capacity and quality of life upon programme completion in adults with asthma. The certainty of evidence relating to maximal exercise capacity was very low to low. Pulmonary rehabilitation appears to confer minimal effect on asthma control, although the certainty of evidence is very low to low. Unclear reporting of study methods and small sample sizes limits our certainty in the overall body of evidence, whilst heterogenous study designs and interventions likely contribute to inconsistent findings across clinical outcomes and studies. There remains considerable scope for future research.

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Conflict of interest statement

CR Osadnik was recipient of a Lung Foundation Australia/Boehringer Ingelheim COPD Research Fellowship during 2016 to 2018 (unrelated to the present work). He has received fees from Novartis for non-promotional speaking engagements (unrelated to the present work).
C Gleeson was supported by an Evidence Synthesis Ireland fellowship.
VM McDonald has participated in educational symposia funded by GlaxoSmithKline, AstraZeneca, and Menarini, and has participated on advisory boards for GlaxoSmithKline, Novartis, AstraZeneca, and Menarini (unrelated to the present work).
AE Holland has received fees from AstraZeneca and Boehringer Ingelheim for non-promotional speaking engagements (unrelated to the present work).
To the best of all authors' knowledge, at the time of submitting this work, none of the named entities have any financial interest in the findings of this review and do not manufacture any such intervention or competing product(s).

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- doi: 10.1002/14651858.CD013485

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Review

Clin Exp Allergy

. 2022 Aug 22.

doi: 10.1111/cea.14220. Online ahead of print.

Airway Autoimmunity, Asthma Exacerbations, and Response to Biologics

Carmen Venegas[1,2], Manali Mukherjee[1,2], Anurag Bhalla[3], Parameswaran Nair[1,2]

Affiliations expand

- PMID: 35993511

- DOI: 10.1111/cea.14220

Abstract

Biologic therapies in asthma are indicated in severe disease and they are directed against specific inflammatory modulators that contribute to the pathogenesis and severity. Currently approved biologics target T2 cytokines (IgE, IL-5, IL-4/IL-13, and TLSP) and have demonstrated efficacy in clinical outcomes such as exacerbation rate and oral corticosteroid dose reductions, blood and airway eosinophil depletion, and lung function improvement. However, a proportion of these patients may show inadequate responses to biologics, with either initial lack of improvement or clinical and functional worsening after an optimal initial response. Exacerbations while on a

biologic may be due to several reason including imprecise identification of the dominant effector pathway contributing to severity, additional inflammatory pathways that are not targeted by the biologic, inaccuracies of the biomarker used to guide therapy, inadequate dosing schedules, intercurrent airway infections, anti-drug neutralizing antibodies, and a novel phenomenon of autoimmune responses in the airways interfering with the effectiveness of the monoclonal antibodies. This review, illustrated using case scenarios, describe the underpinnings of airway autoimmune responses in driving exacerbations while patients are being treated with biologics, device a strategy to evaluate such exacerbations, an algorithm to switch between biologics, and perhaps to consider two biologics concurrently.

Keywords: Severe asthma; airway infections; autoimmunity; biologics; eosinophils; sputum cell counts.

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Review

Eur Respir J

. 2022 Aug 25;60(2):2102782.

doi: 10.1183/13993003.02782-2021. Print 2022 Aug.

Allergen provocation tests in respiratory research: building on 50 years of experience

Gail M Gauvreau[1], Beth E Davis[2], Guy Scadding[3], Louis-Philippe Boulet[4], Leif Bjermer[5], Adam Chaker[6], Donald W Cockcroft[2], Barbro Dahlén[7], Wyste Fokkens[8], Peter Hellings[8], Nikolaos Lazarinis[7], Paul M O'Byrne[9], Ellen Tufvesson[5], Santiago Quirce[10], Maurits Van Maaren[11], Frans H de Jongh[12], Zuzana Diamant[13][14][15]

Affiliations expand

- PMID: 35086834

- PMCID: PMC9403392

- DOI: 10.1183/13993003.02782-2021

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Abstract

The allergen provocation test is an established model of allergic airway diseases, including asthma and allergic rhinitis, allowing the study of allergen-induced changes in respiratory physiology and inflammatory mechanisms in sensitised individuals as well as their associations. In the upper airways, allergen challenge is focused on the clinical and pathophysiological sequelae of the early allergic response, and is applied both as a diagnostic tool and in research settings. In contrast, bronchial allergen challenge has almost exclusively served as a research tool in specialised research settings with a focus on the late asthmatic response and the underlying type 2 inflammation. The allergen-induced late asthmatic response is also characterised by prolonged airway narrowing, increased nonspecific airway hyperresponsiveness and features of airway remodelling including the small airways, and hence allows the study of several key mechanisms and features of asthma. In line with these characteristics, allergen challenge has served as a valued tool to study the cross-talk of the upper and lower airways and in proof-of-mechanism studies of drug development. In recent years, several new insights into respiratory phenotypes and endotypes including the involvement of the upper and small airways, innovative biomarker sampling methods and detection techniques, refined lung function testing as well as targeted treatment options further shaped the applicability of the allergen provocation test in precision medicine. These topics, along with descriptions of subject populations and safety, in line with the updated Global Initiative for Asthma 2021 document, will be addressed in this review.

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Conflict of interest statement

G.M. Gauvreau declares grants from AstraZeneca, Biohaven, Novartis and Genentech; consulting fees from AstraZeneca, Biohaven, Novartis, Sterna Biologicals and Certior Consulting; and payment or honoraria from AstraZeneca, Genzyme and Genentech, all in the 36 months prior to manuscript submission. B.E. Davis declares no competing interests. G. Scadding declares support from ALK-Abelló (provision of active and placebo medication for the GRASS clinical trial referenced in the article) and the Immune Tolerance Network (NIAID, NIH, USA; sponsor of the

GRASS clinical trial) related to the present manuscript; as well as payment for lectures from ALK-Abelló in 2020 and 2021; and support from GlaxoSmithKline for attendance at the 2021 European Academy of Allergy and Clinical Immunology meeting. L-P. Boulet declares research grants for participation in multicentre studies or research projects proposed by the investigator from Amgen, AstraZeneca, GlaxoSmithKline, Merck, Novartis and Sanofi-Regeneron; royalties from UptoDate and Taylor & Francis; lecture fees from AstraZeneca, Covis, GlaxoSmithKline, Novartis, Merck and Sanofi, all in the 36 months prior to manuscript submission; and the following unpaid contributions: Chair of the Global Initiative for Asthma (GINA) Board of Directors; President of the Global Asthma Organisation (Interasma); Member of the Canadian Thoracic Society Respiratory Guidelines Committee; and Laval University Chair on Knowledge Transfer, Prevention and Education in Respiratory and Cardiovascular Health. L. Bjermer declares no competing interests. A. Chaker declares grants or contracts, via Technical University Munich, from EIT Health (European Institute of Technology), BMBF (German Federal Ministry of Education and Research), AstraZeneca, Sanofi-Genzyme, Roche, GlaxoSmithKline and ALK-Abelló; payments or honoraria, via Technical University Munich, from ALK-Abelló, Allergopharma, AstraZeneca, GlaxoSmithKline, Immunotek, Novartis, Regeneron, Sanofi-Genzyme, Leti, Zeller and Bencard; travel reimbursement to attend European Academy of Allergy and Clinical Immunology meetings (2018 and 2021 from the European Academy of Allergy and Clinical Immunology; 2019 from ALK-Abelló); patents, via Technical University Munich, relating to allergen-specific immunotherapy and treatment of SARS-CoV-2 infection; participation, via Technical University Munich, on a data safety monitoring board or advisory board for ALK-Abelló, Allergopharma, AstraZeneca, GlaxoSmithKline, Immunotek, Novartis, Regeneron and Sanofi-Genzyme; and the following unpaid roles: Past Chair of the Allergy, Clinical Immunology and Environmental Medicine Section of the German ENT Society; board member at large of the German Allergy Society (DGAKI); scientific advisor/board member to the German Society for Applied Allergy (AeDA); board member of the Allergen Immuntherapy Interest Group, European Academy of Allergy and Clinical Immunology. D.W. Cockcroft reports the following research grants in the 36 months prior to manuscript submission: research grant Dept of Medicine University of Saskatchewan (Effect of tiotropium on airway response to allergen); Canadian Society of Allergy and Clinical Immunology (The effect of deep inhalation on mannitol responsiveness); AstraZeneca (A phase 2a double blind randomised parallel group placebo controlled multicentre study to evaluate the effect of AZD8154 administered via nebuliser once daily on allergen-induced inflammation in subjects with mild allergic asthma challenged with inhaled allergen); AllerGen NCE (Methacholine challenge comparison of a jet nebuliser and vibrating mesh nebuliser); Novartis (Inhaled CSJ117 in adult asthmatics with mild atopic asthma). B. Dahlén declares payment to their institution in 2004 for a clinical trial by AstraZeneca, related to the present work; and grants to support the Karolinska Severe Asthma Centre from GlaxoSmithKline and Novartis; consulting fees from Teva, AstraZeneca and Sanofi; and payment for lectures from AstraZeneca and Sanofi, all in the 36 months prior to manuscript submission. W. Fokkens declares grants to their institution from ALK, Mylan, Allergy Therapeutics, GlaxoSmithKline, Sanofi, Novartis and Chordate; consulting fees from Sanofi and Bioinspire; payment or honoraria from Sanofi, GlaxoSmithKline and Novartis; and participation on a data safety monitoring board or advisory board for Lyra, Sanofi and GlaxoSmithKline, all in the 36 months prior to manuscript submission; and that they are Secretary General of the European Rhinologic Society. P. Hellings declares no competing interests. N. Lazarinis declares no competing interests. P.M. O'Byrne reports grants from AstraZeneca, GlaxoSmithKline, Biohaven, Merck, Bayer and Novartis; consulting fees from AstraZeneca, Covis, GlaxoSmithKline, Amgen and Novartis; and payment or honoraria from AstraZeneca, Covis and Novartis, all in the 36 months prior to manuscript submission; and that they are a Section Editor of the European Respiratory Journal. E. Tufvesson declares no competing interests. S. Quirce declares consulting fees and payment or honoraria from GlaxoSmithKline, Sanofi and AstraZeneca; and payment or honoraria from Novartis, Chiesi, Mundipharma and Teva, all in the 36 months prior to

manuscript submission. M. Van Maaren declares payment or honoraria from ALK-Abelló, Takeda and CSL Behring, in the 36 months prior to manuscript submission; and an unpaid role as Chairman of the Dutch Society of Allergology and Clinical Immunology since 2020. F. H. de Jongh declares an unpaid role as European Respiratory Society Assessment Director. Z. Diamant reports that until May 2020 they worked as Research Director Respiratory & Allergy at a CRO (QPS-NL), which received funding from biotech and several pharma companies for conduct of phase I–II clinical studies (drug development); and declares consulting fees from ALK-Abelló, AstraZeneca, Antabio, GlaxoSmithKline, HAL Allergy, QPS-NL and Sanofi-Genzyme; and payment or honoraria from BMR, Boehringer Ingelheim, European Forum for Research and Education in Allergy and Airway Diseases (EUFOREA), Merck Sharp & Dohme and Sanofi-Genzyme, all in the 36 months prior to manuscript submission; and unpaid roles as the European Academy of Allergy and Clinical Immunology Asthma Section Chair (2017–2019) and EUFOREA Asthma Expert Panel Chair (since 2020).

- 174 references
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Randomized Controlled Trial

Clin Infect Dis

. 2022 Aug 24;75(1):e473-e481.

doi: 10.1093/cid/ciab813.

Randomized Study of Rivaroxaban vs Placebo on Disease Progression and

Symptoms Resolution in High-Risk Adults With Mild Coronavirus Disease 2019

Jintanat Ananworanich[1], Robin Mogg[1], Michael W Dunne[1], Mohamed Bassyouni[1], Consuela Vera David[2], Erika Gonzalez[3], Taryn Rogalski-Salter[1], Heather Shih[1], Jared Silverman[1], Jeroen Medema[1], Penny Heaton[1]

Affiliations expand

- PMID: 34523673
- PMCID: PMC8522357
- DOI: 10.1093/cid/ciab813

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Abstract

Background: Severe acute respiratory syndrome coronavirus 2 infection may be associated with a prothrombotic state, predisposing patients for a progressive disease course. We investigated whether rivaroxaban, a direct oral anticoagulant factor Xa inhibitor, would reduce coronavirus disease 2019 (COVID-19) progression.

Methods: Adults (N = 497) with mild COVID-19 symptoms and at high risk for COVID-19 progression based on age, body mass index, or comorbidity were randomized 1:1 to either daily oral rivaroxaban 10 mg (N = 246) or placebo equivalent (N = 251) for 21 days and followed to day 35. Primary end points were safety and progression. Absolute difference in progression risk was assessed using a stratified Miettinen and Nurminen method.

Results: The study was terminated after 497 of the target 600 participants were enrolled due to a prespecified interim analysis of the first 200 participants that crossed the futility boundary for the primary efficacy end point in the intent-to-treat population. Enrollees were 85% aged <65 years; 60% female; 27% Hispanic, Black, or other minorities; and 69% with ≥2 comorbidities. Rivaroxaban was well tolerated. Disease progression rates were 46 of 222 (20.7%) in rivaroxaban vs 44 of 222 (19.8%) in placebo groups, with a risk difference of -1.0 (95% confidence interval, -6.4 to 8.4; P = .78).

Conclusions: We did not demonstrate an impact of rivaroxaban on disease progression in high-risk adults with mild COVID-19. There remains a critical public health gap in identifying scalable effective therapies for high-risk people in the outpatient setting to prevent COVID-19 progression.

Keywords: COVID-19; SARS-CoV-2; infection; pneumonia; rivaroxaban.

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- Cited by 5 articles
- 27 references
- 3 figures

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Clin Immunol

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. 2022 Aug 24;109101.

doi: 10.1016/j.clim.2022.109101. Online ahead of print.

5-HT is associated with the dysfunction of regulating T cells in patients with allergic rhinitis

Gui Yang[1], Gaohui Wu[2], Wenkai Yao[3], Li Guan[2], Xiaorui Geng[4], Jiangqi Liu[4], Zhiqiang Liu[4], Liteng Yang[2], Qinmiao Huang[2], Xianhai Zeng[4], Pingchang Yang[5]

Affiliations expand

- PMID: 36029976

- DOI: 10.1016/j.clim.2022.109101

Abstract

The dysfunction of regulating T lymphocytes (Treg) is associated with the pathogenesis of many diseases. Serotonin (5-HT) is capable of interacting with immune cells. The objective of the present study is to shed light on the role of 5-HT in regulating Treg activities. Blood samples were collected from patients with perennial allergic rhinitis (AR). Tregs were isolated from blood samples by magnetic cell sorting. The levels of 5-HT and other cytokines were determined by enzyme-linked immunosorbent assay. The results showed that serum 5-HT levels in patients with AR were higher than in healthy control (HC) subjects. A positive correlation was identified in the data between 5-HT concentrations and AR-related cytokine concentrations in the serum. A negative correlation was found between serum levels of 5-HT and the peripheral frequency of Treg. Exposure to 5-HT enhanced the expression of IL-6 and IL-21 in dendritic (DC) cells. Co-culture of 5-HT-primed DCs with Tregs led to the conversion of Th17 cells. STAT3 blockade efficiently abolished the 5-HT-associated conversion of Th17 cells from Tregs. In summary, patients with AR exhibited higher serum concentrations of 5-HT. 5-HT-primed DCs could convert Tregs to Th17 cells.

Keywords: 5-HT; Allergy; Dendritic cells; Nose; Regulatory T cells.

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Conflict of interest statement

Declaration of Competing Interest None to declare.

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Ecotoxicol Environ Saf

. 2022 Aug 24;243:114005.

doi: 10.1016/j.ecoenv.2022.114005. Online ahead of print.

Nonylphenol exacerbates ovalbumin-induced allergic rhinitis via the TSLP-TSLPR/IL-7R pathway and JAK1/2-STAT3 signaling in a mouse model

Yunxiu Wang[1], Zhiwei Cao[1], He Zhao[1], Zhaowei Gu[2]

Affiliations expand

- PMID: 36029577

- DOI: 10.1016/j.ecoenv.2022.114005

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Abstract

Nonylphenol (NP) can be widely used as a plasticizer, surfactant, antioxidant, textile printing, dyeing additive, and pesticide emulsifier. Animal studies have shown that NP aggravates ovalbumin (OVA)-induced allergic rhinitis (AR); however, the exact mechanism underlying its action has not yet been detailed. This study aimed to explore the aggravation of the AR inflammatory response following NP exposure and its possible mechanism. The AR mouse model was constructed using OVA. Under NP exposure, allergic nasal symptoms were observed, eosinophil infiltration was assessed by Sirius red staining, and the levels of IL-4, IL-5, and IL-13 in nasal mucosa samples were detected using cytometric bead array. The mRNA levels of OX40/OX40L and GATA3 in nasal mucosa were detected by qPCR, and the expression levels of the TSLP and JAK1/2-STAT3 signaling pathway components were also identified. Our results suggest that NP exposure exacerbated allergic nasal symptoms and that eosinophils accumulated in nasal mucosa after OVA challenge. The levels of the typical T helper 2 cytokines, as well as the mRNA levels of OX40/OX40L and GATA3, were elevated in the nasal mucosa of OVA-challenged mice exposed to NP. In addition, NP exposure elevated the TSLP, TSLPR, IL-7R, p-JAK1, p-JAK2, and p-STAT3

levels in the nasal mucosa after OVA stimulation. Overall, the present study suggests NP can exacerbate OVA-induced AR inflammatory responses; furthermore, this aggravating effect of NP may be related to the TSLP-TSLPR/IL-7R and JAK1/2-STAT3 signaling pathways.

Keywords: Allergic rhinitis; Cytokine; Mechanism; Nonylphenol; OX40; OX40L; Th2 cell response.

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Sci Rep

. 2022 Aug 25;12(1):14492.

doi: 10.1038/s41598-022-18734-3.

Scraping nasal cytology in the diagnostics of rhinitis and the comorbidities

Dorota Myszkowska[1], Monika Bazgier[2], Sara Brońska[2], Karol Nowak[3], Joanna Ożga[2], Aleksandra Woźniak[2 3], Andrzej Stanisz[4], Joanna Szaleniec[3]

Affiliations expand

- PMID: 36008516

- PMCID: PMC9403955

- DOI: 10.1038/s41598-022-18734-3

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Abstract

Nasal scraping cytology is a non-invasive tool used in the diagnostics of allergic and non-allergic rhinitis. The study aimed to analyze to what extent the cytological picture of the nasal mucosa coincides with the diagnosis of a given disease, taking into account the content of eosinophils. Retrospective analysis of the cytograms performed in 842 patients was carried out in relation to the disease entities and the content of eosinophils. Significant relationship between the Epith:Infl ratio and the four groups of diseases ($Chi^2 = 9.6488$; $p = .014$) was confirmed. The more intensive inflammation was found, the higher percentage of patients had manifested the increased level of eosinophils (> 1% in the inflammatory cells). The value of 20% of eosinophils in all counted cells corresponds to around 45% of eosinophils in the inflammatory cells in patients with the evident inflammatory picture. Allergic rhinitis presents a different cytological picture regarding the eosinophilic reaction against the background of the inflammation process: the higher degree of inflammation observed, the lower amount of eosinophils detected, with the exception of allergic rhinitis provoked by pollen allergens.

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Conflict of interest statement

The authors declare no competing interests.

- 45 references
- 4 figures

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Clin Exp Immunol

. 2022 Aug 24;uxac074.

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Upregulated expression of substance P and NK1R in blood monocytes and B cells of patients with allergic rhinitis and asthma

Peixuan Han[1], Liping Chen[2], Dong Chen[3], Ruiming Yang[3], Wei Wang[3], Jingyu Liu[3], Shaoheng He[1,3], Huiyun Zhang[1]

Affiliations expand

- PMID: 36001730

- DOI: 10.1093/cei/uxac074

Abstract

Increased expression of substance P (SP) and neurokinin-1 receptor (NK1R) has been noticed in patients with allergic rhinitis (AR) and allergic asthma (AA). However, little is known of the expression of SP and NK1R in monocytes and B cells of AR and AA. In the present study, the expression levels of SP and NK1R were determined by flow cytometry and mouse AR and AA models. The results showed that both percentages of SP + monocytes and SP + B cells, and mean fluorescence intensity (MFI) of SP in monocytes were elevated in the blood of AA and AR combined with AA (ARA) patients. Similarly, the percentages of NK1R + monocytes were elevated in the blood of AR, AA and ARA patients. Allergens artemisia sieversiana wild allergen extract (ASWE), house dust mite extract (HDME) and platanus pollen allergen extract (PPE) increased the expression density of SP molecules (determined by MFI) in an individual monocyte of AR patients. HDME and PPE appeared to enhance SP and NK1R expression in the B cells of ARA and AR patients. In the mouse AR and AA models, the percentages of NK1R + monocytes and B cells were elevated in blood following OVA sensitization and challenge. Knocking out the FcεRI molecule completely abolished the OVA-induced upregulation of expression of NK1R in monocytes and B cells of AA mice. In conclusion, upregulated expressions of SP and NK1R may contribute to the pathogenesis of airway allergy.

Keywords: B cell; NK1R; allergic asthma; allergic rhinitis; monocyte; substance P.

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Review

J Investig Allergol Clin Immunol

. 2022 Aug 24;0.

doi: 10.18176/jiaci.0853. Online ahead of print.

Eosinophilic esophagitis due to aeroallergens: A Systematic Review, update, and our experience

A R Gratacós Gómez[1], E Gómez Torrijos[1]

Affiliations expand

- PMID: 36000828

- DOI: 10.18176/jiaci.0853

Abstract

Eosinophilic esophagitis is a chronic antigen-mediated esophageal disease characterized by symptoms related to esophageal dysfunction and histologically by Th2 inflammation (at least15 eosinophils/high power field) when other secondary systemic and local causes of esophageal eosinophilia are excluded. Though this disease was initially ascribed to a delayed reaction to food allergens, emerging evidence suggests that aeroallergens may also play roles in his pathogenesis and evolution. Some studies support seasonal variation in Eosinophilic esophagitis diagnosis and disease exacerbations about the increase in aeroallergens to which patients are sensitized. It is also known that this disease can be generated after an extensive, identifiable aeroallergen exposure and after treatment with specific immunotherapy with food or aeroallergens. Recently, it has been

postulated that treatment of allergic rhinoconjunctivitis may improve Eosinophilic esophagitis symptoms, though data is limited to case reports and small series. Currently, biomarkers and biologic therapies are not helpful for diagnosis or inducing clinical and histological remission of the disease. Although there are high hopes for dupilumab This review aims to give visibility to the involvement of aeroallergens in the triggering and exacerbation of eosinophilic esophagitis since many of them, in addition to being in the air and being able to inhale them, can also ingest them as part of the food. It is essential to highlight that we must try to discover the cause of the disease since it is crucial for its remission.

Keywords: Aeroallergens; Asthma; Eosinophilic esophagitis; Pollen; Rhinitis.

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Review

Eur Respir J

. 2022 Aug 25;60(2):2102782.

doi: 10.1183/13993003.02782-2021. Print 2022 Aug.

Allergen provocation tests in respiratory research: building on 50 years of experience

Gail M Gauvreau[1], Beth E Davis[2], Guy Scadding[3], Louis-Philippe Boulet[4], Leif Bjermer[5], Adam Chaker[6], Donald W Cockcroft[2], Barbro Dahlén[7], Wyste Fokkens[8], Peter Hellings[8], Nikolaos Lazarinis[7], Paul M O'Byrne[9], Ellen Tufvesson[5], Santiago Quirce[10], Maurits Van Maaren[11], Frans H de Jongh[12], Zuzana Diamant[13 14 15]

Affiliations expand

- PMID: 35086834

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Abstract

The allergen provocation test is an established model of allergic airway diseases, including asthma and allergic rhinitis, allowing the study of allergen-induced changes in respiratory physiology and inflammatory mechanisms in sensitised individuals as well as their associations. In the upper airways, allergen challenge is focused on the clinical and pathophysiological sequelae of the early allergic response, and is applied both as a diagnostic tool and in research settings. In contrast, bronchial allergen challenge has almost exclusively served as a research tool in specialised research settings with a focus on the late asthmatic response and the underlying type 2 inflammation. The allergen-induced late asthmatic response is also characterised by prolonged airway narrowing, increased nonspecific airway hyperresponsiveness and features of airway remodelling including the small airways, and hence allows the study of several key mechanisms and features of asthma. In line with these characteristics, allergen challenge has served as a valued tool to study the cross-talk of the upper and lower airways and in proof-of-mechanism studies of drug development. In recent years, several new insights into respiratory phenotypes and endotypes including the involvement of the upper and small airways, innovative biomarker sampling methods and detection techniques, refined lung function testing as well as targeted treatment options further shaped the applicability of the allergen provocation test in precision medicine. These topics, along with descriptions of subject populations and safety, in line with the updated Global Initiative for Asthma 2021 document, will be addressed in this review.

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Conflict of interest statement

G.M. Gauvreau declares grants from AstraZeneca, Biohaven, Novartis and Genentech; consulting fees from AstraZeneca, Biohaven, Novartis, Sterna Biologicals and Certior Consulting; and payment or honoraria from AstraZeneca, Genzyme and Genentech, all in the 36 months prior to manuscript submission. B.E. Davis declares no competing interests. G. Scadding declares support from ALK-Abelló (provision of active and placebo medication for the GRASS clinical trial referenced in the article) and the Immune Tolerance Network (NIAID, NIH, USA; sponsor of the GRASS clinical trial) related to the present manuscript; as well as payment for lectures from ALK-Abelló in 2020 and 2021; and support from GlaxoSmithKline for attendance at the 2021 European Academy of Allergy and Clinical Immunology meeting. L-P. Boulet declares research grants for participation in multicentre studies or research projects proposed by the investigator from Amgen, AstraZeneca, GlaxoSmithKline, Merck, Novartis and Sanofi-Regeneron; royalties from UptoDate and Taylor & Francis; lecture fees from AstraZeneca, Covis, GlaxoSmithKline, Novartis, Merck and Sanofi, all in the 36 months prior to manuscript submission; and the following unpaid contributions: Chair of the Global Initiative for Asthma (GINA) Board of Directors; President of the Global Asthma Organisation (Interasma); Member of the Canadian Thoracic Society Respiratory

Guidelines Committee; and Laval University Chair on Knowledge Transfer, Prevention and Education in Respiratory and Cardiovascular Health. L. Bjermer declares no competing interests. A. Chaker declares grants or contracts, via Technical University Munich, from EIT Health (European Institute of Technology), BMBF (German Federal Ministry of Education and Research), AstraZeneca, Sanofi-Genzyme, Roche, GlaxoSmithKline and ALK-Abelló; payments or honoraria, via Technical University Munich, from ALK-Abelló, Allergopharma, AstraZeneca, GlaxoSmithKline, Immunotek, Novartis, Regeneron, Sanofi-Genzyme, Leti, Zeller and Bencard; travel reimbursement to attend European Academy of Allergy and Clinical Immunology meetings (2018 and 2021 from the European Academy of Allergy and Clinical Immunology; 2019 from ALK-Abelló); patents, via Technical University Munich, relating to allergen-specific immunotherapy and treatment of SARS-CoV-2 infection; participation, via Technical University Munich, on a data safety monitoring board or advisory board for ALK-Abelló, Allergopharma, AstraZeneca, GlaxoSmithKline, Immunotek, Novartis, Regeneron and Sanofi-Genzyme; and the following unpaid roles: Past Chair of the Allergy, Clinical Immunology and Environmental Medicine Section of the German ENT Society; board member at large of the German Allergy Society (DGAKI); scientific advisor/board member to the German Society for Applied Allergy (AeDA); board member of the Allergen Immuntherapy Interest Group, European Academy of Allergy and Clinical Immunology. D.W. Cockcroft reports the following research grants in the 36 months prior to manuscript submission: research grant Dept of Medicine University of Saskatchewan (Effect of tiotropium on airway response to allergen); Canadian Society of Allergy and Clinical Immunology (The effect of deep inhalation on mannitol responsiveness); AstraZeneca (A phase 2a double blind randomised parallel group placebo controlled multicentre study to evaluate the effect of AZD8154 administered via nebuliser once daily on allergen-induced inflammation in subjects with mild allergic asthma challenged with inhaled allergen); AllerGen NCE (Methacholine challenge comparison of a jet nebuliser and vibrating mesh nebuliser); Novartis (Inhaled CSJ117 in adult asthmatics with mild atopic asthma). B. Dahlén declares payment to their institution in 2004 for a clinical trial by AstraZeneca, related to the present work; and grants to support the Karolinska Severe Asthma Centre from GlaxoSmithKline and Novartis; consulting fees from Teva, AstraZeneca and Sanofi; and payment for lectures from AstraZeneca and Sanofi, all in the 36 months prior to manuscript submission. W. Fokkens declares grants to their institution from ALK, Mylan, Allergy Therapeutics, GlaxoSmithKline, Sanofi, Novartis and Chordate; consulting fees from Sanofi and Bioinspire; payment or honoraria from Sanofi, GlaxoSmithKline and Novartis; and participation on a data safety monitoring board or advisory board for Lyra, Sanofi and GlaxoSmithKline, all in the 36 months prior to manuscript submission; and that they are Secretary General of the European Rhinologic Society. P. Hellings declares no competing interests. N. Lazarinis declares no competing interests. P.M. O'Byrne reports grants from AstraZeneca, GlaxoSmithKline, Biohaven, Merck, Bayer and Novartis; consulting fees from AstraZeneca, Covis, GlaxoSmithKline, Amgen and Novartis; and payment or honoraria from AstraZeneca, Covis and Novartis, all in the 36 months prior to manuscript submission; and that they are a Section Editor of the European Respiratory Journal. E. Tufvesson declares no competing interests. S. Quirce declares consulting fees and payment or honoraria from GlaxoSmithKline, Sanofi and AstraZeneca; and payment or honoraria from Novartis, Chiesi, Mundipharma and Teva, all in the 36 months prior to manuscript submission. M. Van Maaren declares payment or honoraria from ALK-Abelló, Takeda and CSL Behring, in the 36 months prior to manuscript submission; and an unpaid role as Chairman of the Dutch Society of Allergology and Clinical Immunology since 2020. F. H. de Jongh declares an unpaid role as European Respiratory Society Assessment Director. Z. Diamant reports that until May 2020 they worked as Research Director Respiratory & Allergy at a CRO (QPS-NL), which received funding from biotech and several pharma companies for conduct of phase I–II clinical studies (drug development); and declares consulting fees from ALK-Abelló, AstraZeneca, Antabio, GlaxoSmithKline, HAL Allergy, QPS-NL and Sanofi-Genzyme; and payment or honoraria from BMR, Boehringer Ingelheim, European Forum for Research and Education in Allergy and Airway

Diseases (EUFOREA), Merck Sharp & Dohme and Sanofi-Genzyme, all in the 36 months prior to manuscript submission; and unpaid roles as the European Academy of Allergy and Clinical Immunology Asthma Section Chair (2017–2019) and EUFOREA Asthma Expert Panel Chair (since 2020).

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Association of respiratory symptoms and lung function with occupation in the multinational Burden of Obstructive Lung Disease (BOLD) study

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Affiliations expand

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Abstract

Chronic obstructive pulmonary disease has been associated with exposures in the workplace. We aimed to assess the association of respiratory symptoms and lung function with occupation in the Burden of Obstructive Lung Disease study. We analysed cross-sectional data from 28,823 adults (≥40 years) in 34 countries. Eleven occupations were considered and grouped by likelihood of exposure to organic dusts, inorganic dusts and fumes. The association of chronic cough, chronic phlegm, wheeze, dyspnoea, FEV1/FVC and FVC with occupation was assessed, per study site, using multivariable regression. These estimates were then meta-analysed. Sensitivity analyses explored differences between sexes and gross national income (GNI). Overall, working in settings with potentially high exposure to dusts or fumes was associated with respiratory symptoms but not lung function differences. The most common occupation was farming. Compared to people not working in any of the 11 considered occupations, those who were farmers for ≥20 years were more likely to have chronic cough (OR=1.52, 95%CI 1.19-1.94), wheeze (OR=1.37, 95%CI 1.16-1.63), and dyspnoea (OR=1.83, 95%CI 1.53-2.20), but not lower FVC (β=0.02L, 95%CI -0.02L to 0.06L) or lower FEV_1/FVC (β=0.04%, 95%CI -0.49% to 0.58%). Some findings differed by sex and GNI. In summary, at a population level, the occupational exposures considered in this study do not appear to be major determinants of differences in lung function, although they associate with more respiratory symptoms. As not all work settings were included in this study, respiratory surveillance should still be encouraged among high-risk dusty and fume job workers, especially in low- and middle-income countries.

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Early Diagnosis in Cerebellar Ataxia, Neuropathy, Vestibular Areflexia Syndrome (CANVAS) by Focusing on Major Clinical Clues: Beyond Ataxia and Vestibular Impairment

Laurent Magy[1,2], Pauline Chazelas[1,3], Laurence Richard[1,2], Nathalie Deschamps[2], Simon Frachet[1,2], Jean-Michel Vallat[1,2], Corinne Magdelaine[1,3], Frédéric Favreau[1,3], Flavien Bessaguet[1], Anne-Sophie Lia[1,3,4], Mathilde Duchesne[1,5]

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Abstract

CANVAS, a rare disorder responsible for late-onset ataxia of autosomal recessive inheritance, can be misdiagnosed. We investigated a series of eight patients with sensory neuropathy and/or an unexplained cough, who appeared to suffer from CANVAS, and we emphasized the clinical clues for early diagnosis. Investigations included clinical and routine laboratory analyses, skin biopsy, nerve biopsy and molecular genetics. The eight patients had clinical and/or laboratory evidence of sensory neuronopathy. All but one had neuropathic pain that had started in an asymmetric fashion in two patients. A chronic cough was a prominent feature in our eight patients and had started years before neuropathic symptoms in all but one. The course of the disease was slow, and ataxia remained mild in all. Five patients were initially thought to have immune-mediated sensory neuronopathy and received immunotherapy. Skin biopsies showed a near complete and non-length-dependent loss of intraepidermal nerve fibers. Moreover, nerve biopsy findings suggested a prominent involvement of small myelinated and unmyelinated fibers. The burden of CANVAS extends far beyond cerebellar ataxia and vestibular manifestations. Indeed, our study shows that a chronic cough and neuropathic pain may represent a major source of impairment in these patients and should not be overlooked to allow an early diagnosis and prevent unnecessary immunotherapy.

Keywords: CANVAS; chronic cough; early diagnosis; sensory neuropathy.

Conflict of interest statement

The authors declare no conflict of interest.

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JAMA Pediatr

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Clinical Features and Burden of Postacute Sequelae of SARS-CoV-2 Infection in Children and Adolescents

Suchitra Rao[1], Grace M Lee[2], Hanieh Razzaghi[3], Vitaly Lorman[3], Asuncion Mejias[4], Nathan M Pajor[5], Deepika Thacker[6], Ryan Webb[3], Kimberley Dickinson[3], L Charles Bailey[3], Ravi Jhaveri[7], Dimitri A Christakis[8,9], Tellen D Bennett[1], Yong Chen[10], Christopher B Forrest[3]

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Abstract

Importance: The postacute sequelae of SARS-CoV-2 infection (PASC) has emerged as a long-term complication in adults, but current understanding of the clinical presentation of PASC in children is limited.

Objective: To identify diagnosed symptoms, diagnosed health conditions, and medications associated with PASC in children.

Design, setting and participants: This retrospective cohort study used electronic health records from 9 US children's hospitals for individuals younger than 21 years who underwent antigen or reverse transcriptase-polymerase chain reaction (RT-PCR) testing for SARS-CoV-2 between March 1, 2020, and October 31, 2021, and had at least 1 encounter in the 3 years before testing.

Exposures: SARS-CoV-2 positivity by viral test (antigen or RT-PCR).

Main outcomes and measures: Syndromic (symptoms), systemic (conditions), and medication PASC features were identified in the 28 to 179 days following the initial test date. Adjusted hazard ratios (aHRs) were obtained for 151 clinically predicted PASC features by contrasting viral test-positive groups with viral test-negative groups using proportional hazards models, adjusting for site, age, sex, testing location, race and ethnicity, and time period of cohort entrance. The incidence proportion for any syndromic, systemic, or medication PASC feature was estimated in the 2 groups to obtain a burden of PASC estimate.

Results: Among 659 286 children in the study sample, 348 091 (52.8%) were male, and the mean (SD) age was 8.1 (5.7) years. A total of 59 893 (9.1%) tested positive by viral test for SARS-CoV-2, and 599 393 (90.9%) tested negative. Most were tested in outpatient testing facility settings (322 813 [50.3%]) or office settings (162 138 [24.6%]). The most common syndromic, systemic, and medication features were loss of taste or smell (aHR, 1.96; 95% CI, 1.16-3.32), myocarditis (aHR,

3.10; 95% CI, 1.94-4.96), and cough and cold preparations (aHR, 1.52; 95% CI, 1.18-1.96), respectively. The incidence of at least 1 systemic, syndromic, or medication feature of PASC was 41.9% (95% CI, 41.4-42.4) among viral test-positive children vs 38.2% (95% CI, 38.1-38.4) among viral test-negative children, with an incidence proportion difference of 3.7% (95% CI, 3.2-4.2). A higher strength of association for PASC was identified in those cared for in the intensive care unit during the acute illness phase, children younger than 5 years, and individuals with complex chronic conditions.

Conclusions and relevance: In this large-scale, exploratory study, the burden of pediatric PASC that presented to health systems was low. Myocarditis was the most commonly diagnosed PASC-associated condition. Acute illness severity, young age, and comorbid complex chronic disease increased the risk of PASC.

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