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COPD

Respir Res

. 2022 Aug 15;23(1):207.

doi: 10.1186/s12931-022-02128-0.

Impact of chronic obstructive pulmonary disease on short-term outcome in patients with ST-elevation myocardial infarction during COVID-19 pandemic: insights from the international multicenter ISACS-STEMI registry

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Bakraceski[33], Filippo Zilio[34], Luca Donazzan[35], Adriaan Kraaijeveld[36], Gennaro Galasso[37], Arpad Lux[38], Lucia Marinucci[39], Vincenzo Guiducci[40], Maurizio Menichelli[41], Alessandra Scoccia[42], Aylin Hatice Yamac[43], Kadir Ugur Mert[44], Xacobe Flores Rios[45], Tomas Kovarnik[46], Michal Kidawa[47], Josè Moreu[48], Vincent Flavien[49], Enrico Fabris[50], Iñigo Lozano Martínez-Luengas[51], Marco Boccalatte[52], Francisco Bosa Ojeda[53], Carlos Arellano-Serrano[54], Gianluca Caiazzo[55], Giuseppe Cirrincione[56], Hsien-Li Kao[57], Juan Sanchis Forés[58], Luigi Vignali[59], Helder Pereira[60], Stephane Manzo[61], Santiago Ordoñez[62], Alev Arat Özkan[63], Bruno Scheller[64], Heidi Lehtola[65], Rui Teles[66], Christos Mantis[67], Ylitalo Antti[68], João A Brum Silveira[69], Rodrigo Zoni[70], Ivan Bessonov[71], Stefano Savonitto[72], George Kochiadakis[73], Dimitrios Alexopoulos[74], Carlos E Uribe[75], John Kanakakis[76], Benjamin Faurie[77], Gabriele Gabrielli[78], Alejandro Gutierrez Barrios[79], Juan Pablo Bachini[80], Alex Rocha[81], Frankie Chor-Cheung Tam[82], Alfredo Rodriguez[83], Antonia Anna Lukito[84], Veauthyelau Saint-Joy[85], Gustavo Pessah[86], Andrea Tuccillo[16], Giuliana Cortese[87], Guido Parodi[88], Mohamed Abed Bouraghda[89], Elvin Kedhi[90], Pablo Lamelas[62], Harry Suryapranata[91], Monica Verdoia[92]

Affiliations expand

- PMID: 35971173

- DOI: 10.1186/s12931-022-02128-0

Abstract

Background: Chronic obstructive pulmonary disease (COPD) is projected to become the third cause of mortality worldwide. COPD shares several pathophysiological mechanisms with cardiovascular disease, especially atherosclerosis. However, no definite answers are available on the prognostic role of COPD in the setting of ST elevation myocardial infarction (STEMI), especially during COVID-19 pandemic, among patients undergoing primary angioplasty, that is therefore the aim of the current study.

Methods: In the ISACS-STEMI COVID-19 registry we included retrospectively patients with STEMI treated with primary percutaneous coronary intervention (PCI) between March and June of 2019 and 2020 from 109 high-volume primary PCI centers in 4 continents.

Results: A total of 15,686 patients were included in this analysis. Of them, 810 (5.2%) subjects had a COPD diagnosis. They were more often elderly and with a more pronounced cardiovascular risk profile. No preminent procedural dissimilarities were noticed except for a lower proportion of dual antiplatelet therapy at discharge among COPD patients (98.9% vs. 98.1%, P = 0.038). With regards to short-term fatal outcomes, both in-hospital and 30-days mortality occurred more frequently among COPD patients, similarly in pre-COVID-19 and COVID-19 era. However, after adjustment for main baseline differences, COPD did not result as independent predictor for in-hospital death (adjusted OR [95% CI] = 0.913[0.658-1.266], P = 0.585) nor for 30-days mortality (adjusted OR [95% CI] = 0.850 [0.620-1.164], P

= 0.310). No significant differences were detected in terms of SARS-CoV-2 positivity between the two groups.

Conclusion: This is one of the largest studies investigating characteristics and outcome of COPD patients with STEMI undergoing primary angioplasty, especially during COVID pandemic. COPD was associated with significantly higher rates of in-hospital and 30-days mortality. However, this association disappeared after adjustment for baseline characteristics. Furthermore, COPD did not significantly affect SARS-CoV-2 positivity.

Trial registration number: NCT04412655 (2nd June 2020).

Keywords: COPD; Mortality; STEMI.

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- 40 references

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Respirology

. 2022 Aug 15.

doi: 10.1111/resp.14339. Online ahead of print.

Effect of chronic mucus hypersecretion on treatment responses to inhaled therapies in patients with chronic obstructive pulmonary disease: Post hoc analysis of the IMPACT trial

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Affiliations expand

- PMID: 35970518

- DOI: 10.1111/resp.14339

Abstract

Background and objective: Chronic mucus hypersecretion (CMH) is a clinical phenotype of COPD. This exploratory post hoc analysis assessed relationship between CMH status and treatment response in IMPACT.

Methods: Patients were randomized to once-daily fluticasone furoate/umeclidinium/vilanterol (FF/UMEC/VI) 100/62.5/25 µg, FF/VI 100/25 µg or UMEC/VI 62.5/25 µg and designated CMH+ if they scored 1/2 in St George's Respiratory Questionnaire (SGRQ) questions 1 and 2. Endpoints assessed by baseline CMH status included on-treatment exacerbation rates, change from baseline in trough forced expiratory volume in 1 second, SGRQ total score, COPD Assessment Test (CAT) score, proportion of SGRQ and CAT responders at Week 52 and safety.

Results: Of 10,355 patients in the intent-to-treat population, 10,250 reported baseline SGRQ data (CMH+: 62% [n = 6383]). FF/UMEC/VI significantly ($p < 0.001$) reduced on-treatment moderate/severe exacerbation rates versus FF/VI and UMEC/VI in CMH+ (rate ratio: 0.87 and 0.72) and CMH- patients (0.82 and 0.80). FF/UMEC/VI significantly ($p < 0.05$) reduced on-treatment severe exacerbation rates versus UMEC/VI in CMH+ (0.62) and CMH- (0.74) subgroups. Similar improvements in health status and lung function with FF/UMEC/VI were observed, regardless of CMH status. In CMH+ patients, FF/VI significantly ($p < 0.001$) reduced on-treatment moderate/severe and severe exacerbation rates versus UMEC/VI (0.83 and 0.70).

Conclusion: FF/UMEC/VI had a favourable benefit: risk profile versus dual therapies irrespective of CMH status. The presence of CMH did not influence treatment response or exacerbations, lung function and/or health status. However, CMH did generate differences when dual therapies were compared and the impact of CMH should be considered in future trial design.

Keywords: COPD; chronic mucus hypersecretion; chronic obstructive pulmonary disease; clinical outcomes; single-inhaler triple therapy.

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- 29 references

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Respir Med

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. 2022 Aug 9;201:106944.

doi: 10.1016/j.rmed.2022.106944. Online ahead of print.

Stability of distinct symptom experiences in patients with chronic obstructive pulmonary disease (COPD)

Vivi L Christensen[1], Tone Rustøen[2], Magne Thoresen[3], Are M Holm[4], Signe B Bentsen[5]

Affiliations expand

- PMID: 35970058

- DOI: 10.1016/j.rmed.2022.106944

Abstract

Purpose: This study aimed to examine reclassification rates among classes of chronic obstructive pulmonary disease (COPD) patients based on their distinct symptom experiences and to assess how these subgroups differed in symptom scores and health-

related quality of life (HRQoL) outcomes over one year. Moreover, we wished to assess how these subgroups differed in demographic and clinical characteristics at 12 months.

Patients and methods: This is a follow-up study of 267 patients with moderate, severe, and very severe COPD. Based on their distinct symptom experiences using the Memorial Symptom Assessment Scale (MSAS), three subgroups (i.e., "high", "intermediate", and "low") were identified at baseline. In the present study, transitions between the subgroups at three, six, nine, and 12 months were investigated and calculated as reclassification rates. Differences among the subgroups in symptom scores and HRQoL at each time point and demographic and clinical characteristics at 12 months were evaluated using analysis of variance with post hoc comparisons.

Results: Almost 65% were still in the "high" class after 12 months. At 12 months, pairwise comparisons for respiratory function measurements were not significantly different. Compared to the "intermediate" and "low" class, patients in the "high" class were more likely to be women and had significantly more comorbidities, reported a significantly higher number of symptoms at all time points, and worse HRQoL scores.

Conclusion: Our findings suggest that the pattern of a high symptom burden in COPD is consistent over time. The patients' individual symptom experiences should be the primary focus of treatment.

Keywords: Latent class analyses; Longitudinal; Stability; Subgroups; Symptom burden.

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Ann Am Thorac Soc

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Diffusing Capacity and Mortality in Chronic Obstructive Pulmonary Disease

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Affiliations expand

- PMID: 35969416

- DOI: 10.1513/AnnalsATS.202203-226OC

Abstract

Rationale: Chronic Obstructive Pulmonary Disease (COPD) mortality risk is often estimated using the BODE index (including body mass index, forced expiratory volume in one second (FEV1), dyspnea score, and six-minute walk distance). Diffusing capacity (DLCO) is a potential predictor of mortality that reflects physiology distinct from that in the BODE index. **Objectives**: This study evaluated DLCO as a predictor of mortality using participants from the COPDGene study. **Methods**: We performed time-to-event analyses of individuals with COPD (former/current smokers with FEV1/FVC <0.7) and DLCO measurements from the COPDGene Phase 2 visit. Cox proportional hazard methods were used to model survival, adjusting for age, sex, pack-years, smoking status, BODE index, computed tomography (CT) percent emphysema (low attenuation areas <-950 Hounsfield units), CT airway wall thickness, and history of cardiovascular or kidney diseases. C-statistics for models with DLCO and BODE score were used to compare discriminative accuracy. **Results**: Of 2329 participants, 378(16.8%) died during the follow-up period (median 4.9 years). In adjusted analyses, for every 10% decrease in DLCO %predicted, mortality increased by 29% (Hazard ratio 1.29, 95% CI 1.17 - 1.41, p<0.001). When compared to other clinical predictors, DLCO %predicted performed similarly to BODE (C-statistic DLCO 0.68, BODE 0.70), and the addition of DLCO to BODE improved its discriminative accuracy (C-statistic 0.71). **Conclusions**: Diffusing capacity, a measure of gas transfer, strongly predicted all-cause mortality in individuals with COPD, independent of BODE index and CT evidence of emphysema and airway wall thickness. These findings support inclusion of DLCO in prognostic models for COPD.

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Lancet

. 2022 Aug 13;400(10351):471-473.

doi: 10.1016/S0140-6736(22)01383-6.

Biologic therapies for asthma in underserved populations

Rachel S Kelly[1], Scott T Weiss[2]

Affiliations expand

- PMID: 35964595

- DOI: 10.1016/S0140-6736(22)01383-6

No abstract available

Conflict of interest statement

STW reports royalty payments from UpToDate for advice on the epidemiology of asthma and chronic obstructive pulmonary disease and is an unpaid board member of Histolix, a digital pathology company. RSK declares no competing interests.

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Sci Rep

. 2022 Aug 13;12(1):13789.

doi: 10.1038/s41598-022-17959-6.

Longitudinal follow-up of the asthma status in a French-Canadian cohort

Marie-Eve Lavoie[1,2], Jolyane Meloche[1,2], Anne-Marie Boucher-Lafleur[1,2], Paul Bégin[3], Charles Morin[3], Louis-Philippe Boulet[4], Anne-Marie Madore[1,2], Catherine Laprise[5,6,7]

Affiliations expand

- PMID: 35963877

- PMCID: PMC9376060

- DOI: 10.1038/s41598-022-17959-6

Abstract

Asthma affects 340 million people worldwide and varies in time. Twenty years ago, in Canada, the Saguenay-Lac-Saint-Jean asthma family cohort was created to study the genetic and environmental components of asthma. This study is a follow-up of 125 participants of this cohort to explore the appearance, persistence, and progression of asthma over 10-20 years. Participants answered a clinical standardized questionnaire. Lung function was assessed (forced expiratory volume in 1 s, forced vital capacity, bronchial reversibility, and methacholine bronchoprovocation), skin allergy testing was performed, blood samples were obtained (immunoglobulin E, white blood cell counts) and phenotypes were compared between recruitment and follow-up. From the participants without asthma at recruitment, 12% developed a phenotype of adult-onset asthma with the presence of risk factors, such as atopy, high body mass index, and exposure to smoking. A decrease of PC_{20} values in this group was observed and a decrease in the FEV_1/FVC ratio in all groups. Also, 7% of individuals with asthma at recruitment developed chronic obstructive pulmonary disease, presenting risk factors at recruitment, such as moderate-to-severe bronchial hyperresponsiveness, exposure to smoking, and asthma. This study allowed a better interpretation of the evolution of asthma. Fine phenotypic characterization is the first step for meaningful genetic and epigenetic studies.

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Conflict of interest statement

The authors declare no competing interests.

- 36 references
- 3 figures

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Cost Eff Resour Alloc

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doi: 10.1186/s12962-022-00377-w.

Cost-effectiveness of integrated disease management for high risk, exacerbation prone, patients with chronic obstructive pulmonary disease in a primary care setting

Andrew D Scarffe[1], Christopher J Licskai[2,3,4], Madonna Ferrone[4,5], Kevin Brand[6], Kednapa Thavorn[7,8], Doug Coyle[8,9]

Affiliations expand

- PMID: 35962399
- PMCID: PMC9373353
- DOI: 10.1186/s12962-022-00377-w

Abstract

Background: We evaluate the cost-effectiveness of the 'Best Care' integrated disease management (IDM) program for high risk, exacerbation prone, patients with chronic obstructive pulmonary disease (COPD) compared to usual care (UC) within a primary care setting from the perspective of a publicly funded health system (i.e., Ontario, Canada).

Methods: We conducted a model-based, cost-utility analysis using a Markov model with expected values of costs and outcomes derived from a Monte-Carlo Simulation with 5000 replications. The target population included patients started in GOLD II with a starting age of 68 years in the trial-based analysis. Key input parameters were based on a randomized control trial of 143 patients (i.e., UC (n = 73) versus IDM program (n = 70)). Results were shown as incremental cost per quality-adjusted life year (QALY) gained.

Results: The IDM program for high risk, exacerbation prone, patients is dominant in comparison with the UC group. After one year, the IDM program demonstrated cost savings and improved QALYs (i.e., UC was dominated by IDM) with a positive net-benefit of $5360 (95% CI: ($5175, $5546) based on a willingness to pay of $50,000 (CAN) per QALY.

Conclusions: This study demonstrates that the IDM intervention for patients with COPD in a primary care setting is cost-effective in comparison to the standard of care. By demonstrating the cost-effectiveness of IDM, we confirm that investment in the delivery of evidence based best practices in primary care delivers better patient outcomes at a lower cost than UC.

Keywords: COPD; Chronic obstructive pulmonary disease; Cost-effectiveness; Cost-utility; Integrated disease management; Primary care.

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Conflict of interest statement

None to declare.

- 53 references
- 5 figures

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J Appl Physiol (1985)

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. 2022 Aug 11.

doi: 10.1152/japplphysiol.00352.2022. Online ahead of print.

Physiological Predictors of Morbidity and Mortality in COPD: The Relative Importance of Reduced Inspiratory Capacity and Inspiratory Muscle Strength

Devin B Phillips[1], Matthew D James[1], Conor J D O'Donnell[1], Sandra G Vincent[1], Katherine A Webb[1], Juan Pablo de-Torres[1], J Alberto Neder[1], Denis E O'Donnell[1]

Affiliations expand

- PMID: 35952349

- DOI: 10.1152/japplphysiol.00352.2022

Abstract

Low resting inspiratory capacity (IC) and low maximal inspiratory pressure (MIP) have previously been linked to exertional dyspnea, exercise limitation and poor survival in chronic obstructive pulmonary disease (COPD). The interaction and relative contributions of these two related variables to important clinical outcomes are unknown. The objective of the current study was to examine the interaction between resting IC and MIP (both % predicted), exertional dyspnea, exercise capacity and long-term survival in patients with COPD. Two hundred and eighty-five patients with mild to advanced COPD completed standard lung function testing and a cycle cardiopulmonary exercise test. Multiple regression determined predictors of the exertional dyspnea-ventilation slope and peak oxygen uptake ($\dot{V}O_{2peak}$). Cox regression determined predictors of 10-year mortality. IC was associated with the dyspnea-ventilation slope (standardized β=-0.44, $p<0.001$), while MIP was excluded from the regression model ($p=0.713$). IC and MIP were included in the final model to predict $\dot{V}O_{2peak}$. However, the standardized β was greater for IC (0.49) than MIP (0.22). After adjusting for age, sex, body mass index, cardiovascular risk, airflow obstruction and diffusing capacity, resting IC was independently associated with 10-year all-cause mortality (hazard ratio=1.25, confidence interval$_{5\text{-}95\%}$=1.16-1.34, $p<0.001$), while MIP was excluded from the final model (all $p=0.829$). Low resting IC was consistently linked to

heightened dyspnea intensity, low $\dot{V}O_{2peak}$ and worse survival in COPD even after accounting for airway obstruction, inspiratory muscle strength, and diffusing capacity. These results support the use of resting IC as an important physiological biomarker closely linked to key clinical outcomes in COPD.

Keywords: COPD; dyspnea; exercise capacity; inspiratory capacity; inspiratory muscle weakness.

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Review

Eur Respir Rev

. 2022 Aug 10;31(165):220059.

doi: 10.1183/16000617.0059-2022. Print 2022 Sep 30.

Air pollution as an early determinant of COPD

Zhuyi Lu[1], Patrice Coll[2], Bernard Maitre[1 3], Ralph Epaud[1 4], Sophie Lanone[5]

Affiliations expand

- PMID: 35948393

- DOI: 10.1183/16000617.0059-2022

Free article

Abstract

COPD is a progressive and debilitating disease often diagnosed after 50 years of age, but more recent evidence suggests that its onset could originate very early on in life. In this context, exposure to air pollution appears to be a potential contributor. Although the potential role of air pollution as an early determinant of COPD is emerging, knowledge gaps still remain, including an accurate qualification of air pollutants (number of pollutants quantified and exact composition) or the "one exposure-one disease" concept, which might limit the current understanding. To fill these gaps, improvements in the field are needed, such as the use of atmosphere simulation chambers able to realistically reproduce the complexity of air pollution, consideration of the exposome, as well as improving exchanges between paediatricians and adult lung specialists to take advantage of reciprocal expertise. This review should lead to a better understanding of the current knowledge on air pollution as an early determinant of COPD, as well as identify the existing knowledge gaps and opportunities to fill them. Hopefully, this will lead to better prevention strategies to scale down the development of COPD in future generations.

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Conflict of interest statement

Conflict of interest: None of the authors have any conflict of interest to declare.

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Editorial

Eur Respir J

. 2022 Aug 10;60(2):2200898.

doi: 10.1183/13993003.00898-2022. Print 2022 Aug.

Cystic fibrosis transmembrane conductance regulator (CFTR): a missing link between smoking and chronic airway diseases?

Pierre-Régis Burgel[1,2], Nicolas Roche[3,2]

Affiliations expand

- PMID: 35948348

- DOI: 10.1183/13993003.00898-2022

No abstract available

Conflict of interest statement

Conflict of Interest: P-R. Burgel reports grants from Boehringer Ingelheim, GSK and Vertex; consulting fees and lecture honoraria from Boehringer Ingelheim, GSK, AstraZeneca, Vertex, Chiesi, Pfizer, Novartis, Zambon and Insmed; outside the submitted work. N. Roche reports grants from Boehringer Ingelheim, Novartis, GSK and Pfizer; consulting fees from Boehringer Ingelheim, GSK, AstraZeneca, Sanofi, Chiesi, Pfizer, Novartis and Teva; lecture honoraria from Boehringer Ingelheim, GSK, AstraZeneca, Sanofi, Chiesi, Pfizer, Novartis, Teva and Zambon; outside the submitted work.

Comment on

- *C FTR* variants are associated with chronic bronchitis in smokers.
  Saferali A, Qiao D, Kim W, Raraigh K, Levy H, Diaz AA, Cutting GR, Cho MH, Hersh CP; NHLBI TransOmics in Precision Medicine (TOPMed).Eur Respir J. 2022 Aug 10;60(2):2101994. doi: 10.1183/13993003.01994-2021. Print 2022 Aug.PMID: 34996830

SUPPLEMENTARY INFO

Publication types, MeSH terms, Substances, Supplementary conceptsexpand

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J Acad Consult Liaison Psychiatry

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doi: 10.1016/j.jaclp.2022.07.008. Online ahead of print.

Association Between COPD Severity, Exacerbation Risk, and Anxiety and Depression Symptoms in the SPIROMICS Cohort

Jacob R Weiss[1], Ryan Serdenes[2], Uchechukwu Madtha[3], Huaqing Zhao[4], Victor Kim[5], Jahaira Lopez-Pastrana[6], Michelle N Eakin[7], Jacqueline O'Toole[8], Christopher B Cooper[9], Prescott Woodruff[10], Richard E Kanner[11], Jerry A Krishnan[12], Anand S Iyer[13], David Couper[14], Mary F Morrison[15]

Affiliations expand

- PMID: 35948252

- DOI: 10.1016/j.jaclp.2022.07.008

Abstract

Introduction: Chronic obstructive pulmonary disease (COPD) is a common, progressive lung disease that often manifests with psychiatric symptoms. Despite this, patients with COPD are not routinely screened for anxiety and depression, which substantially contribute to COPD-related morbidity. We hypothesized that COPD participants with more severe respiratory symptoms and at higher risk of COPD exacerbations are more likely to experience clinically significant anxiety and depression.

Methods: To test this hypothesis, we used baseline data from the Subpopulations and Intermediate Outcome Measures In COPD Study (SPIROMICS) cohort to examine ever smokers with COPD across Global Initiative for Obstructive Lung Disease (GOLD) disease severity groups. Multivariable logistic regression models were used to calculate odds ratios for clinically significant anxiety and depression for each GOLD group, which was compared to the ever smokers without COPD control group. Odds ratios were adjusted for subject demographics, medical comorbidities, and substance use covariates, and comparisons were completed using two-tailed tests.

Results: Of the 2,664 subjects studied, 784 (29.4%) had clinically significant anxiety and 497 (18.7%) had clinically significant depression. In the multivariable analysis, high pulmonary symptom groups, groups B and D, had increased adjusted odds of clinically significant anxiety (group B: AOR 1.28, $p=0.03$; group D: AOR 1.95, $p<0.0001$) and depression (group B: AOR 2.09, $p<0.0001$; group D: AOR 3.04, $p<0.0001$). GOLD group D, the high pulmonary symptom and high COPD exacerbation risk group, had the greatest risk of both anxiety and depression among the GOLD groups.

Conclusion: High COPD symptom severity, even in the absence of elevated COPD exacerbation risk, is associated with clinically significant anxiety and depression. Our separate analyses of anxiety and depression symptoms in a large, multisite, national cohort is unique within the literature and has important treatment implications for COPD patients. Our findings also highlight the utility of screening patients with high COPD symptom severity for anxiety and depression.

Keywords: Anxiety; Chronic Obstructive Pulmonary Disease; Depression; Global Initiative for Obstructive Lung Disease groups; Hospital Anxiety and Depression Scale; Subpopulations and Intermediate Outcome Measures In COPD Study.

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J Clin Sleep Med

. 2022 Aug 10.

doi: 10.5664/jcsm.10210. Online ahead of print.

Therapy for insomnia with chronic obstructive pulmonary disease: a randomized trial of components

Mary Kapella[1], Alana Steffen[1], Bharati Prasad[1,2], Franco Laghi[3,4], Sachin Vispute[5], Gretchen Kemner[1,6], Celso Teixeira[7], Tara Peters[1], Jeehye Jun[1], Julie Law[1], David Carley[1]

Affiliations expand

- PMID: 35946416

- DOI: 10.5664/jcsm.10210

Abstract

Study objectives: To determine efficacy and mechanisms of cognitive behavioral therapy for insomnia (CBT-I) and COPD education (COPD-ED) on clinical outcomes in adults with concurrent COPD and insomnia.

Methods: We conducted a 2 × 2 factorial study to test the impact of CBT-I and COPD-ED delivered alone or in combination on severity of insomnia and fatigue, sleep and dyspnea. Participants were randomized to one of 4 groups. Group1: CBT-I+Attention Control (AC, health videos, N=27). Group 2: COPD-ED+AC, N=28. Group 3: CBT-I+COPD-ED, N=27 and Group 4, AC only, N=27. Participants received 6, 75-minute weekly sessions. Dependent variables included insomnia severity, sleep by actigraphy, fatigue and dyspnea measured at baseline, immediately post-intervention, and at 3-months post-intervention. Presumed mediators of intervention effects included beliefs and attitudes about sleep, self-efficacy for sleep and COPD, and emotional function.

Results: COPD patients (FEV1% predicted 67±24% (mean ± SD), age 65±8 years, with insomnia participated in the study. Insomnia and sleep improved more in patients who received CBT-I than in those who did not, an effect that was sustained at 3 months post-intervention and mediated by beliefs and attitudes about sleep. CBT-I was associated with clinically important improvements in fatigue and dyspnea. When CBT-I and COPD-ED were concurrently administered, effects on insomnia, fatigue and dyspnea were attenuated.

Conclusions: CBT-I produced significant and sustained decreases in insomnia, sleep and clinically important improvement in fatigue and dyspnea. Combination of CBT-I and COPD-ED reduce CBT-I's effectiveness. Further research is needed to understand the mechanisms associated with effects of insomnia therapy on multiple symptoms in COPD.

Clinical trial registration: Registry: ClinicalTrials.gov; Title: A Behavioral Therapy for Insomnia Co-existing with COPD; Identifier: NCT01973647.

Keywords: chronic obstructive pulmonary disease; cognitive behavioral therapy; dyspnea; fatigue; insomnia; sleep.

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BMC Pulm Med

. 2022 Aug 9;22(1):306.

doi: 10.1186/s12890-022-02100-0.

Development and validation of a prognostic nomogram among patients with acute exacerbation of chronic obstructive pulmonary disease in intensive care unit

Jiang-Chen Peng[#1], Wen-Wen Gong[#2], Yan Wu[2], Tian-Yi Yan[3], Xiao-Yan Jiang[4,5]

Affiliations expand

- PMID: 35945553
- PMCID: PMC9364535
- DOI: 10.1186/s12890-022-02100-0

Free PMC article

Abstract

Background: Acute exacerbation of Chronic Obstructive Pulmonary Disease (AECOPD) contributes significantly to mortality among patients with COPD in Intensive care unit (ICU). This study aimed to develop a nomogram to predict 30-day mortality among AECOPD patients in ICU.

Methods: In this retrospective cohort study, we extracted AECOPD patients from Medical Information Mart for Intensive Care III (MIMIC-III) database. Multivariate logistic regression based on Akaike information criterion (AIC) was used to establish the nomogram. Internal validation was performed by a bootstrap resampling approach with 1000 replications. The discrimination and calibration of the nomogram were evaluated by Harrell's concordance index (C-index) and Hosmer-Lemeshow (HL) goodness-of-fit test. Decision curve analysis (DCA) was performed to evaluate its clinical application.

Results: A total of 494 patients were finally included in the study with a mean age of 70.8 years old. 417 (84.4%) patients were in the survivor group and 77 (15.6%) patients were in the non-survivor group. Multivariate logistic regression analysis based on AIC included age, pO_2, neutrophil-to-lymphocyte ratio (NLR), prognostic nutritional index (PNI), invasive mechanical ventilation and vasopressor use to construct the nomogram. The adjusted C-

index was 0.745 (0.712, 0.778) with good calibration (HL test, P = 0.147). The Kaplan-Meier survival curves revealed a significantly lower survival probability in the high-risk group than that in the low-risk group (P < 0.001). DCA showed that nomogram was clinically useful.

Conclusion: The nomogram developed in this study could help clinicians to stratify AECOPD patients and provide appropriate care in clinical setting.

Keywords: 30-day mortality; AECOPD; Intensive care unit; Nomogram; Predictive model.

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Conflict of interest statement

None of the authors has any potential conflicts of interest related to this article to declare and the results of this report have been produced, analyzed, and interpreted without any outside participation.

- 33 references
- 5 figures

SUPPLEMENTARY INFO

MeSH termsexpand

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J Med Screen

. 2022 Aug 9;9691413221117685.

doi: 10.1177/09691413221117685. Online ahead of print.

The prevalence of comorbidity in the lung cancer screening population: A systematic review and meta-analysis

Anas Almatrafi[1,2], Owen Thomas[1], Matthew Callister[3], Rhian Gabe[4], Rebecca J Beeken[1,5], Richard Neal[1,6]

Affiliations expand

- PMID: 35942779

- DOI: 10.1177/09691413221117685

Abstract

Objective: Comorbidity is associated with adverse outcomes for all lung cancer patients, but its burden is less understood in the context of screening. This review synthesises the prevalence of comorbidities among lung cancer screening (LCS) candidates and summarises the clinical recommendations for screening comorbid individuals.

Methods: We searched MEDLINE, EMBASE, EBM Reviews, and CINAHL databases from January 1990 to February 2021. We included LCS studies that reported a prevalence of comorbidity, as a prevalence of a particular condition, or as a summary score. We also summarised LCS clinical guidelines that addressed comorbidity or frailty for LCS as a secondary objective for this review. Meta-analysis was used with inverse-variance weights obtained from a random-effects model to estimate the prevalence of selected comorbidities.

Results: We included 69 studies in the review; seven reported comorbidity summary scores, two reported performance status, 48 reported individual comorbidities, and 12 were clinical guideline papers. The meta-analysis of individual comorbidities resulted in an estimated prevalence of 35.2% for hypertension, 23.5% for history of chronic obstructive pulmonary disease (COPD) (10.7% for severe COPD), 16.6% for ischaemic heart disease (IHD), 13.1% for peripheral vascular disease (PVD), 12.9% for asthma, 12.5% for diabetes, 4.5% for bronchiectasis, 2.2% for stroke, and 0.5% for pulmonary fibrosis.

Conclusions: Comorbidities were highly prevalent in LCS populations and likely to be more prevalent than in other cancer screening programmes. Further research on the burden of comorbid disease and its impact on screening uptake and outcomes is needed. Identifying individuals with frailty and comorbidities who might not benefit from screening should become a priority in LCS research.

Keywords: Lung cancer; comorbidity; frailty; low-dose computed tomography; screening.

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15

J Med Chem

. 2022 Aug 11;65(15):10233-10250.

doi: 10.1021/acs.jmedchem.2c00609. Epub 2022 Jul 28.

Discovery of Clinical Candidate CHF-6366: A Novel Super-soft Dual Pharmacology Muscarinic Antagonist and β_2 Agonist (MABA) for the Inhaled Treatment of Respiratory Diseases

Laura Carzaniga[1], Ian D Linney[2], Andrea Rizzi[1], Maurizio Delcanale[1], Wolfgang Schmidt[2], Christopher K Knight[2], Fiorella Pastore[3], Daniela Miglietta[3], Chiara Carnini[4], Nicola Cesari[5], Benedetta Riccardi[5], Valentina Mileo[6], Luca Venturi[6], Elisa Moretti[6], Wesley P Blackaby[2], Riccardo Patacchini[4], Alessandro Accetta[1], Matteo Biagetti[1], Franco Bassani[3], Marina Tondelli[3], Annalisa Murgo[3], Loredana Battipaglia[7], Gino Villetti[3], Paola Puccini[5], Silvia Catinella[6], Maurizio Civelli[8], Fabio Rancati[1]

Affiliations expand

- PMID: 35901125

- DOI: 10.1021/acs.jmedchem.2c00609

Abstract

The development of molecules embedding two distinct pharmacophores acting as muscarinic antagonists and β_2 agonists (MABAs) promises to be an excellent opportunity to reduce formulation issues and boost efficacy through cross-talk and allosteric interactions. Herein, we report the results of our drug discovery campaign aimed at improving the therapeutic index of a previous MABA series by exploiting the super soft-drug concept. The incorporation of a metabolic liability, stable at the site of administration but undergoing rapid systemic metabolism, to generate poorly active and quickly eliminated fragments was pursued. Our SAR studies yielded MABA **29**, which demonstrated a balanced in vivo profile up to 24 h, high instability in plasma and the liver, as well as sustained exposure in the lung. In vitro safety and non-GLP toxicity studies supported the nomination of **29** (CHF-6366) as a clinical candidate, attesting to the successful development of a novel super-soft MABA compound.

SUPPLEMENTARY INFO

MeSH terms, Substancesexpand

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JCI Insight

. 2022 Aug 8;7(15):e155581.

doi: 10.1172/jci.insight.155581.

IL-36 receptor agonist and antagonist imbalance drives neutrophilic inflammation in COPD

Jonathan R Baker[1], Peter S Fenwick[1], Carolin K Koss[2], Harriet B Owles[1], Sarah L Elkin[3], Jay Fine[4], Matthew Thomas[2], Karim C El Kasmi[2], Peter J Barnes[1], Louise E Donnelly[1]

Affiliations expand

- PMID: 35763349

- DOI: 10.1172/jci.insight.155581

Free article

Abstract

Current treatments fail to modify the underlying pathophysiology and disease progression of chronic obstructive pulmonary disease (COPD), necessitating alternative therapies. Here, we show that COPD subjects have increased IL-36γ and decreased IL-36 receptor antagonist (IL-36Ra) in bronchoalveolar and nasal fluid compared with control subjects. IL-36γ is derived from small airway epithelial cells (SAEC) and is further induced by a viral mimetic, whereas IL-36Ra is derived from macrophages. IL-36γ stimulates release of the neutrophil chemoattractants CXCL1 and CXCL8, as well as elastolytic matrix metalloproteinases (MMPs) from small airway fibroblasts (SAF). Proteases released from COPD neutrophils cleave and activate IL-36γ, thereby perpetuating IL-36 inflammation. Transfer of culture media from SAEC to SAF stimulated release of CXCL1, which was inhibited by exogenous IL-36Ra. The use of a therapeutic antibody that inhibits binding to the IL-36R attenuated IL-36γ-driven inflammation and cellular crosstalk. We have demonstrated a mechanism for the amplification and propagation of neutrophilic inflammation in COPD and have shown that blocking this cytokine family via a IL-36R neutralizing antibody could be a promising therapeutic strategy in the treatment of COPD.

Keywords: COPD; Cell Biology; Cellular immune response; Cytokines; Pulmonology.

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Am J Respir Crit Care Med

. 2022 Aug 15;206(4):427-439.

doi: 10.1164/rccm.202110-2241OC.

Lung Microbiota and Metabolites Collectively Associate with Clinical Outcomes in Milder Stage Chronic Obstructive Pulmonary Disease

Siddharth S Madapoosi[1], Charmion Cruickshank-Quinn[2], Kristopher Opron[1], John R Erb-Downward[1], Lesa A Begley[1], Gen Li[3], Igor Barjaktarevic[4], R Graham Barr[5 6], Alejandro P Comellas[7], David J Couper[8], Christopher B Cooper[4], Christine M Freeman[1], MeiLan K Han[1], Robert J Kaner[9], Wassim Labaki[1], Fernando J Martinez[9], Victor E Ortega[10], Stephen P Peters[10], Robert Paine[11], Prescott Woodruff[12], Jeffrey L Curtis[1 13], Gary B Huffnagle[1 14], Kathleen A Stringer[15], Russell P Bowler[16 17], Charles R Esther Jr[18 19], Nichole Reisdorph[2], Yvonne J Huang[1 20], SPIROMICS Research Group

Collaborators, Affiliations expand

- PMID: 35536732

- DOI: 10.1164/rccm.202110-2241OC

Abstract

Rationale: Chronic obstructive pulmonary disease (COPD) is variable in its development. Lung microbiota and metabolites collectively may impact COPD pathophysiology, but relationships to clinical outcomes in milder disease are unclear. **Objectives:** Identify components of the lung microbiome and metabolome collectively associated with clinical markers in milder stage COPD. **Methods:** We analyzed paired microbiome and metabolomic data previously characterized from bronchoalveolar lavage fluid in 137 participants in the SPIROMICS (Subpopulations and Intermediate Outcome Measures in COPD Study), or (GOLD [Global Initiative for Chronic Obstructive Lung Disease Stage 0-2).

Datasets used included *1*) bacterial 16S rRNA gene sequencing; *2*) untargeted metabolomics of the hydrophobic fraction, largely comprising lipids; and *3*) targeted metabolomics for a panel of hydrophilic compounds previously implicated in mucoinflammation. We applied an integrative approach to select features and model 14 individual clinical variables representative of known associations with COPD trajectory (lung function, symptoms, and exacerbations). **Measurements and Main Results:** The majority of clinical measures associated with the lung microbiome and metabolome collectively in overall models (classification accuracies, >50%, $P < 0.05$ vs. chance). Lower lung function, COPD diagnosis, and greater symptoms associated positively with *Streptococcus*, *Neisseria*, and *Veillonella*, together with compounds from several classes (glycosphingolipids, glycerophospholipids, polyamines and xanthine, an adenosine metabolite). In contrast, several *Prevotella* members, together with adenosine, 5'-methylthioadenosine, sialic acid, tyrosine, and glutathione, associated with better lung function, absence of COPD, or less symptoms. Significant correlations were observed between specific metabolites and bacteria ($P_{adj} < 0.05$). **Conclusions:** Components of the lung microbiome and metabolome in combination relate to outcome measures in milder COPD, highlighting their potential collaborative roles in disease pathogenesis.

Keywords: bronchoscopy; chronic obstructive pulmonary disease; lung function; metabolomics.

Comment in

- Microbiomics-focused Data Integration: A Fresh Solve for the Rubik's Cube of Endophenotyping?
  Narayana JK, Tsaneva-Atanasova K, Chotirmall SH.Am J Respir Crit Care Med. 2022 Aug 15;206(4):365-368. doi: 10.1164/rccm.202205-0860ED.PMID: 35584334 No abstract available.

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Am J Respir Crit Care Med

. 2022 Aug 15;206(4):417-426.

doi: 10.1164/rccm.202108-1943OC.

Endotyping Chronic Obstructive Pulmonary Disease, Bronchiectasis, and the "Chronic Obstructive Pulmonary Disease-Bronchiectasis Association"

Jeffrey T-J Huang[1], Erin Cant[2], Holly R Keir[2], Alun K Barton[1], Elena Kuzmanova[1], Morven Shuttleworth[2], Jennifer Pollock[2], Simon Finch[2], Eva Polverino[3], Mathieu Bottier[2], Alison J Dicker[1], Amelia Shoemark[2], James D Chalmers[2]

Affiliations expand

- PMID: 35436182

- DOI: 10.1164/rccm.202108-1943OC

Abstract

Rationale: Bronchiectasis and chronic obstructive pulmonary disease (COPD) are two disease entities with overlapped clinical features, and codiagnosis frequently occurs (termed the "COPD-bronchiectasis association"). **Objectives:** To investigate the sputum microbiome and proteome in patients with bronchiectasis, COPD, and the COPD-bronchiectasis association with the aim of identifying endotypes that may inform treatment. **Methods:** Sputum microbiome and protein profiling were carried out using 16S rRNA amplicon sequencing and a label-free proteomics workflow, respectively, in a cohort comprising patients with COPD (n = 43), bronchiectasis (n = 30), and the COPD-bronchiectasis association (n = 48). Results were validated in an independent cohort of 91 patients (n = 28-31 each group) using targeted measurements of inflammatory markers, mucins, and bacterial culture. **Measurements and Main Results:** Principal component analysis of sputum microbiome and protein profiles showed a partial separation between the COPD and the "COPD-bronchiectasis association" group. Further analyses revealed that

patients with the "COPD-bronchiectasis association" had a higher abundance of proteobacteria, higher expression of mucin-5AC and proteins from the "neutrophil degranulation" pathway compared to those with COPD. In contrast, patients with COPD had an elevated expression of mucin-5B and several peptidase inhibitors, higher abundance of common commensal taxa, and a greater microbiome diversity. The profiles of "COPD-bronchiectasis association" and bronchiectasis groups were largely overlapping. Five endotypes were proposed with differential inflammatory, mucin, and microbiological features. The key features related to the "COPD-bronchiectasis association" were validated in an independent cohort. **Conclusions:** Neutrophilic inflammation, differential mucin expression, and Gram-negative infection are dominant traits in patients with the "COPD-bronchiectasis association."

Keywords: COPD; bronchiectasis; endotype; microbiome; proteome; sputum; the “COPD–bronchiectasis association”.

Comment in

- Microbiomics-focused Data Integration: A Fresh Solve for the Rubik's Cube of Endophenotyping?
  Narayana JK, Tsaneva-Atanasova K, Chotirmall SH.Am J Respir Crit Care Med. 2022 Aug 15;206(4):365-368. doi: 10.1164/rccm.202205-0860ED.PMID: 35584334 No abstract available.

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Thorax

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. 2022 Aug 15;thoraxjnl-2022-219142.

doi: 10.1136/thorax-2022-219142. Online ahead of print.

Tobacco industry ownership of pharmaceutical companies: an international survey of people with respiratory disease

Ruth Tal-Singer[1,2], Linda Walsh[3], Tim Deuby[3], Angela Shaughnessy[3], Karin F Hoth[4], Victor Gascon[2], Susanne Hintringer[2], Nicole Hass[2,5], Tonya Winders[2], Lucy Morgan[6], Byron Thomashow[3], David M Mannino[3]

Affiliations expand

- PMID: 35970539

- DOI: 10.1136/thorax-2022-219142

Abstract

The 2021 purchase of the respiratory pharmaceutical company Vectura by Phillip Morris International has been criticised by the public health and medical community, as a conflict of interest, with little input to date, from the patient community or the public. To address this gap, the COPD Foundation, along with global partners, surveyed 1196 people with chronic respiratory disease. 70% were bothered by a tobacco company making an inhaler to treat lung conditions and 48% reported that they would want to switch inhalers if they knew that a tobacco company made or sold their inhaler devices. Patients care about who makes the therapies used to treat their diseases.

Keywords: Asthma; Bronchiectasis; COPD epidemiology; Inhaler devices; Tobacco and the lung; Tobacco control.

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Conflict of interest statement

Competing interests: RT-S is a retiree and shareholder of GlaxoSmithKline. She is a non-executive board member of ENA Respiratory and reports holding share options. She also reports personal fees from Vocalis Health, ENA Respiratory, Teva, and Immunomet. TW is President & CEO of Allergy & Asthma Network in the USA. She serves as President of GAAPP. Both AAN and GAAPP receive funding for unbranded disease awareness, education and advocacy from Aimmune, ALK, Amgen, AZ, DBV, GSK, Novartis, Sanofi/Regeneron, Viatris, TEVA. TW personally reports speaker & advisor fees from Aimmune, ALK, Amgen, AZ, DBV, Novartis and Sanofi/Regeneron. BT reports consultancy to GlaxosmithKline and participation at an advisory board for Boehringer Ingelheim. DMM is a former employee and current shareholder of GlaxoSmithKline and receives royalties from Up to Date. He is also a consultant to GlaxoSmithKline, Medical Director of the COPD Foundation, and an expert witness for people suing the Tobacco Industry. LW, TD, AS, KFH, VG, SH, NH and LM report no conflicts.

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J Allergy Clin Immunol Pract

. 2022 Aug 12;S2213-2198(22)00809-1.

doi: 10.1016/j.jaip.2022.08.007. Online ahead of print.

Differences between early and late-onset asthma: role of comorbidities on symptom control

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Affiliations expand

- PMID: 35970446

- DOI: 10.1016/j.jaip.2022.08.007

Abstract

Background: Asthma can present in early childhood or de novo in adulthood. Our understanding of the burden of comorbidities in adult asthmatic patients stratified by age of onset is incomplete.

Objectives: To evaluate how different comorbidities would affect symptom control in two distinct groups of patients with early and late onset asthma (EOA and LOA). To explore whether reported comorbidities associate with lung function and inflammatory parameters.

Methods: We conducted a cross-sectional study of 175 adult asthmatics (57.5±17.1 years) recruited at our University Asthma Clinic. We defined EOA as asthma onset <12 years; LOA as onset >40 years. The primary outcome was symptom control and the main comorbidities evaluated were rhinitis, GERD, obesity, cardiovascular and bronchiectasis. Multivariable regression analysis was used to identify potential predictors of poor control in EOA and LOA.

Results: 77/175 (44%) subjects had EOA, 98 (56%) had LOA and comorbidities had a differential impact in the two groups. Rhinitis was more frequent in EOA (76 vs 53%;p=0.02) and was associated with uncontrolled asthma (p<0.001), reduced FEV_1/FVC (p=0.01), increased eosinophils (p=0.003) and total IgE (p<0.01). Conversely, in LOA rhinitis was associated with more controlled asthma and higher FEV_1/FVC (both p<0.01). In EOA only IgE levels were directly related to blood eosinophils (r=0.42;p<0.001) and inversely to FEV1/FVC (r=- 0.35;p=0.002). Obesity was present in 20% of patients in both groups, but only in LOA obesity was associated to uncontrolled disease (p=0.009), reduced FEV_1/FVC (p=0.009) and blood neutrophils (p=0.03). In multivariable regression analysis rhinitis in EOA and obesity in LOA were the risk factor most closely associated with poor control; GERD, cardiovascular and bronchiectasis did not affect control.

Conclusions: Early-onset persistent and late-onset asthma are distinct phenotypes, with different underlying inflammatory patterns and different comorbidities which impact on symptom control.

Keywords: Age of onset; asthma control; comorbidities; inflammation; lung function.

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Review

Am J Med Sci

. 2022 Aug 12;S0002-9629(22)00331-7.

doi: 10.1016/j.amjms.2022.07.007. Online ahead of print.

Severe eosinophilic granulomatosis with polyangiitis responding to a combination of rituximab and mepolizumab

Georgios Tsioulos[1], Dimitris Kounatidis[2], Natalia G Vallianou[3], Nektarios Koufopoulos[1], Pelagia Katsimbri[1], Anastasia Antoniadou[1]

Affiliations expand

- PMID: 35970248

- DOI: 10.1016/j.amjms.2022.07.007

Abstract

Eosinophilic granulomatosis with polyangiitis (EGPA), formerly known as Churg-Strauss Syndrome, is a multisystem antineutrophil cytoplasmic antibody (ANCA) positive vasculitis, characterized by the presence of chronic rhinosinusitis, asthma and prominent peripheral blood eosinophilia. Although the most commonly involved organ is the lung, followed by the skin, EGPA can affect any organ system. Herein, we present the complicated case of an 18-year-old male patient with severe life-threatening EGPA, with central nervous system, cardiac and gasterointestinal involvement, which was resistant to initial treatment with glucocorticoids and cyclophosphamide. The patient responded well, achieving complete remission after the addition of rituximab and mepolizumab to glucocorticoids and cyclophosphamide.

Keywords: Eosinophilic Granulomatosis with Polyangiitis; cyclophosphamide, mepolizumab; glucocorticoids; rituximab.

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Conflict of interest statement

Conflict of Interest Statement There are no conflict sof interest regarding this manuscript.

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Lancet

. 2022 Aug 13;400(10351):502-511.

doi: 10.1016/S0140-6736(22)01198-9.

Mepolizumab for urban children with exacerbation-prone eosinophilic asthma in the USA (MUPPITS-2): a randomised, double-blind, placebo-controlled, parallel-group trial

Daniel J Jackson[1], Leonard B Bacharier[2], Peter J Gergen[3], Lisa Gagalis[3], Agustin Calatroni[4], Stephanie Wellford[4], Michelle A Gill[5], Jeffrey Stokes[5], Andrew H Liu[6], Rebecca S Gruchalla[7], Robyn T Cohen[8], Melanie Makhija[9], Gurjit K Khurana Hershey[10], George T O'Connor[8], Jacqueline A Pongracic[9], Michael G Sherenian[11], Katherine Rivera-Spoljaric[12], Edward M Zoratti[13], Stephen J Teach[14], Meyer Kattan[15], Cullen M Dutmer[16], Haejin Kim[13], Carin Lamm[17], William J Sheehan[14], R Max Segnitz[18], Kimberly A Dill-McFarland[18], Cynthia M Visness[4], Patrice M Becker[3], James E Gern[19], Christine A Sorkness[20], William W Busse[21], Matthew C Altman[22], US National Institute of Allergy and Infectious Disease's Inner City Asthma Consortium

Affiliations expand

- PMID: 35964610

- DOI: 10.1016/S0140-6736(22)01198-9

Abstract

Background: Black and Hispanic children living in urban environments in the USA have an excess burden of morbidity and mortality from asthma. Therapies directed at the eosinophilic phenotype reduce asthma exacerbations in adults, but few data are available in children and diverse populations. Furthermore, the molecular mechanisms that underlie exacerbations either being prevented by, or persisting despite, immune-based therapies are not well understood. We aimed to determine whether mepolizumab, added to guidelines-based care, reduced the number of asthma exacerbations during a 52-week period compared with guidelines-based care alone.

Methods: This is a randomised, double-blind, placebo-controlled, parallel-group trial done at nine urban medical centres in the USA. Children and adolescents aged 6-17 years, who lived in socioeconomically disadvantaged neighbourhoods and had exacerbation-prone asthma (defined as ≥two exacerbations in the previous year) and blood eosinophils of at least 150 cells per μL were randomly assigned 1:1 to mepolizumab (6-11 years: 40 mg; 12-17 years: 100 mg) or placebo injections once every 4 weeks, plus guideline-based care, for 52 weeks. Randomisation was done using a validated automated system. Participants, investigators, and the research staff who collected outcome measures remained masked to group assignments. The primary outcome was the number of asthma exacerbations that were treated with systemic corticosteroids during 52 weeks in the intention-to-treat population. The mechanisms of treatment response were assessed by study investigators using nasal transcriptomic modular analysis. Safety was assessed in the intention-to-treat population. This trial is registered with ClinicalTrials.gov, NCT03292588.

Findings: Between Nov 1, 2017, and Mar 12, 2020, we recruited 585 children and adolescents. We screened 390 individuals, of whom 335 met the inclusion criteria and were enrolled. 290 met the randomisation criteria, were randomly assigned to mepolizumab (n=146) or placebo (n=144), and were included in the intention-to-treat analysis. 248 completed the study. The mean number of asthma exacerbations within the 52-week study period was 0·96 (95% CI 0·78-1·17) with mepolizumab and 1·30 (1·08-1·57) with placebo (rate ratio 0·73; 0·56-0·96; p=0·027). Treatment-emergent adverse events occurred in 42 (29%) of 146 participants in the mepolizumab group versus 16 (11%) of 144 participants in the placebo group. No deaths were attributed to mepolizumab.

Interpretation: Phenotype-directed therapy with mepolizumab in urban children with exacerbation-prone eosinophilic asthma reduced the number of exacerbations.

Funding: US National Institute of Allergy and Infectious Diseases and GlaxoSmithKline.

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Conflict of interest statement

Declaration of interests All authors, with the exceptions of PMB, LG, and PJG, report grants from the US National Institutes of Health (NIH)–NIAID during the study. CMV, AC, SW, LG, PJG, and PMB have nothing to disclose outside of the submitted work. LBB reports personal fees from GlaxoSmithKline, Genentech–Novartis, Teva, Boehringer Ingelheim, AstraZeneca, WebMD–Medscape, Merck, DBV Technologies, Circassia, Sanofi–Regeneron, and Vectura, all outside the submitted work. GTO reports personal fees from AstraZeneca and grant funding from Janssen Pharmaceuticals, outside the submitted work. CMD reports personal fees from Horizon Therapeutics and Enzyvant Therapeutics, outside the submitted work. MCA reports personal fees from Sanofi–Regeneron, outside the submitted work. WWB reports personal fees from Boston Scientific, Novartis, GlaxoSmithKline, Genentech, Sanofi–Genzyme, AstraZeneca, Teva, Regeneron, and Elsevier, outside the submitted work. MAG reports an honorarium for and support for travel to the 2017 American Academy of Allergy, Asthma & Immunology meeting during this study and monetary compensation from the American Academy of Pediatrics for her work teaching the biannual Pediatrics board review course: PREP The Course. GKKH reports grants from Adare during the study. DJJ reports personal fees from Novartis, Pfizer, Regeneron, AstraZeneca, Sanofi, and Vifor Pharma; grants and personal fees from GlaxoSmithKline; and grants from NIH–US National Heart, Lung, and Blood Institute (NHLBI), all outside the submitted work. MK reports personal fees from Regeneron, outside the submitted work. RSG reports government employment from the Center for Biologics Evaluation and Research and personal fees from Consulting Massachusetts Medical Society, outside the submitted work. AHL reports personal fees from Phadia ThermoFisher as consulting honoraria; grants and non-financial support from ResMed–Propeller Health; non-financial support from Revenio; grants and personal fees from Avillion; and personal fees from Labcorp, all outside the submitted work. SJT reports grants from NIH–Eunice Kennedy Shriver National Institute of Child Health and Human Development, NIH–NHLBI, and EJF Philanthropies and personal fees from Uptodate, all outside the submitted work. JAP reports provision of study drug for another asthma research study from GlaxoSmithKline and Boehringer Ingelheim and provision of study drug for another asthma research study and for food allergy research studies from Genentech–Novartis, all outside the submitted work. All other authors declare no competing interests.

SUPPLEMENTARY INFO

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Lancet

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. 2022 Aug 13;400(10351):471-473.

doi: 10.1016/S0140-6736(22)01383-6.

Biologic therapies for asthma in underserved populations

Rachel S Kelly[1], Scott T Weiss[2]

Affiliations expand

- PMID: 35964595

- DOI: 10.1016/S0140-6736(22)01383-6

No abstract available

Conflict of interest statement

STW reports royalty payments from UpToDate for advice on the epidemiology of asthma and chronic obstructive pulmonary disease and is an unpaid board member of Histolix, a digital pathology company. RSK declares no competing interests.

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Sci Rep

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. 2022 Aug 13;12(1):13789.

doi: 10.1038/s41598-022-17959-6.

Longitudinal follow-up of the asthma status in a French-Canadian cohort

Marie-Eve Lavoie[1,2], Jolyane Meloche[1,2], Anne-Marie Boucher-Lafleur[1,2], Paul Bégin[3], Charles Morin[3], Louis-Philippe Boulet[4], Anne-Marie Madore[1,2], Catherine Laprise[5,6,7]

Affiliations expand

- PMID: 35963877
- PMCID: PMC9376060
- DOI: 10.1038/s41598-022-17959-6

Abstract

Asthma affects 340 million people worldwide and varies in time. Twenty years ago, in Canada, the Saguenay-Lac-Saint-Jean asthma family cohort was created to study the genetic and environmental components of asthma. This study is a follow-up of 125 participants of this cohort to explore the appearance, persistence, and progression of asthma over 10-20 years. Participants answered a clinical standardized questionnaire. Lung function was assessed (forced expiratory volume in 1 s, forced vital capacity, bronchial reversibility, and methacholine bronchoprovocation), skin allergy testing was performed, blood samples were obtained (immunoglobulin E, white blood cell counts) and phenotypes were compared between recruitment and follow-up. From the participants without asthma at recruitment, 12% developed a phenotype of adult-onset asthma with the presence of risk factors, such as atopy, high body mass index, and exposure to smoking. A decrease of PC_{20} values in this group was observed and a decrease in the FEV_1/FVC ratio in all groups. Also, 7% of individuals with asthma at recruitment developed chronic obstructive pulmonary disease, presenting risk factors at recruitment, such as moderate-to-severe bronchial hyperresponsiveness, exposure to smoking, and asthma. This study allowed a better interpretation of the evolution of asthma. Fine phenotypic characterization is the first step for meaningful genetic and epigenetic studies.

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Conflict of interest statement

The authors declare no competing interests.

- 36 references

- 3 figures

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J Allergy Clin Immunol

. 2022 Aug 10;S0091-6749(22)01049-1.

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Human PD-1 agonist treatment alleviates neutrophilic asthma by reprogramming T cells

Doumet Georges Helou[1], Christine Quach[1], Marshall Fung[1], Jacob D Painter[1], Benjamin P Hurrell[1], Yong-Hwee Eddie Loh[2], Emily Howard[1], Pedram Shafiei-Jahani[1], Pejman Soroosh[3], Arlene H Sharpe[4], Omid Akbari[5]

Affiliations expand

- PMID: 35963455

- DOI: 10.1016/j.jaci.2022.07.022

Abstract

Background: Neutrophilic asthma is associated with disease severity and corticosteroid insensitivity. Novel therapies are required to manage this life-threatening asthma phenotype. Programmed cell death protein-1 (PD-1) is a key homeostatic modulator of the immune response for T cell effector functions.

Objective: Our aim here was to investigate the role of PD-1 in the regulation of acute neutrophilic inflammation in a murine model of airway hyperreactivity (AHR).

Methods: House dust mite was used to induce and compare neutrophilic AHR in wild-type and PD-1 knockout mice. Then the therapeutic potential of a human PD-1 agonist was tested in a humanized

mouse model in which the PD-1 extracellular domain is entirely humanized. Single-cell RNA sequencing and flow cytometry were mainly used to investigate molecular and cellular mechanisms.

Results: PD-1 was highly induced on pulmonary T cells in our inflammatory model. PD-1 deficiency was associated with an increased neutrophilic AHR and high recruitment of inflammatory cells to the lungs. Consistently, PD-1 agonist treatment dampened AHR, decreased neutrophil recruitment, and modulated cytokine production in a humanized PD-1 mouse model. Mechanistically, we demonstrated at the transcriptional and protein levels that the inhibitory effect of PD-1 agonist is associated with the reprogramming of pulmonary effector T cells that showed decreased number and activation.

Conclusion: PD-1 agonist treatment is efficient in dampening neutrophilic AHR and lung inflammation in a preclinical humanized mouse model.

Keywords: Neutrophilic asthma; PD-1 agonist; T cells; humanized mice; lung inflammation.

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Cost Eff Resour Alloc

. 2022 Aug 12;20(1):39.

doi: 10.1186/s12962-022-00377-w.

Cost-effectiveness of integrated disease management for high risk, exacerbation prone, patients with chronic obstructive pulmonary disease in a primary care setting

Andrew D Scarffe[1], Christopher J Licskai[2,3,4], Madonna Ferrone[4,5], Kevin Brand[6], Kednapa Thavorn[7,8], Doug Coyle[8,9]

Affiliations expand

- PMID: 35962399
- PMCID: PMC9373353
- DOI: 10.1186/s12962-022-00377-w

Abstract

Background: We evaluate the cost-effectiveness of the 'Best Care' integrated disease management (IDM) program for high risk, exacerbation prone, patients with chronic obstructive pulmonary disease (COPD) compared to usual care (UC) within a primary care setting from the perspective of a publicly funded health system (i.e., Ontario, Canada).

Methods: We conducted a model-based, cost-utility analysis using a Markov model with expected values of costs and outcomes derived from a Monte-Carlo Simulation with 5000 replications. The target population included patients started in GOLD II with a starting age of 68 years in the trial-based analysis. Key input parameters were based on a randomized control trial of 143 patients (i.e., UC (n = 73) versus IDM program (n = 70)). Results were shown as incremental cost per quality-adjusted life year (QALY) gained.

Results: The IDM program for high risk, exacerbation prone, patients is dominant in comparison with the UC group. After one year, the IDM program demonstrated cost savings and improved QALYs (i.e., UC was dominated by IDM) with a positive net-benefit of $5360 (95% CI: ($5175, $5546) based on a willingness to pay of $50,000 (CAN) per QALY.

Conclusions: This study demonstrates that the IDM intervention for patients with COPD in a primary care setting is cost-effective in comparison to the standard of care. By demonstrating the cost-effectiveness of IDM, we confirm that investment in the delivery of evidence based best practices in primary care delivers better patient outcomes at a lower cost than UC.

Keywords: COPD; Chronic obstructive pulmonary disease; Cost-effectiveness; Cost-utility; Integrated disease management; Primary care.

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Conflict of interest statement

None to declare.

- 53 references
- 5 figures

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J Allergy Clin Immunol Pract

. 2022 Aug 9;S2213-2198(22)00799-1.

doi: 10.1016/j.jaip.2022.07.029. Online ahead of print.

Economic impact of anti-IL-5 agents in patients with severe eosinophilic asthma: a population-based cohort study

Paola Faverio[1], Matteo Monzio Compagnoni[2], Raffaella Ronco[2], Matteo Franchi[2], Matteo Della Zoppa[3], Giulia Bonaiti[4], Martina Bonifazi[5], Federico Mei[5], Fabrizio Luppi[4], Alberto Pesci[4], Giovanni Corrao[2]

Affiliations expand

- PMID: 35961616

- DOI: 10.1016/j.jaip.2022.07.029

No abstract available

Keywords: anti-IL-5 agents; benralizumab; economic impact; eosinophilic asthma; mepolizumab; severe asthma.

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Fundam Clin Pharmacol

. 2022 Aug 12.

doi: 10.1111/fcp.12823. Online ahead of print.

ImmunoModulatory Effect of IL-35 Gene Transfected Mesenchymal Stem Cells on Allergic Asthma

Xiang-Hua Bao[1], Feng Gao[2], Seyyed Shamsadin Athari[3], Hongqun Wang[4]

Affiliations expand

- PMID: 35959714

- DOI: 10.1111/fcp.12823

Abstract

Asthma is a common respiratory disease which has no definitive treatment at now. Immune response shifting from T helper (Th)1 to the Th2 is a main problem in asthma, and immunomodulation can help to control asthma. IL-35 and Mesenchymal Stem Cells (MSCs) have regulatory effect on the immune system, and may have the ability to control asthma pathology. After culturing MSCs, expression vector of IL-35 (pUNO1-mIL35elasti) was transduced to the MSCs and then, asthmatic mice were treated with MSCs, MSCs-vector, MSCs-vector-IL-35 and no treatment. AHR, levels of the cytokines, total and OVA specific immunoglobulin (Ig)E, LTB4, and LTC4 were measured. Lung tissue histopathology was also done. MSCs were successfully transduced by pUNO1-mIL35elasti vector, and IL-35 was produced in transduced cells. AHR, levels of the Cytokines, IgEs, LTs, goblet cell hyperplasia, mucus secretion, peribronchial and perivascular inflammation were controlled by MSCs therapy. In MSCs-IL-35 group, these controls were stronger than MSCs without IL-35 group. MSCs had strong effect on control of asthma. Transfected MSCs by expressing IL-35 gene could significantly better control allergic asthma symptoms than MSCs without IL-35. In the future, identification of the IL-35 mechanism of action would be useful to improve cytokine-cell based therapies.

Keywords: Cell therapy; Cytokine; Gene therapy; Immunopathology; Immunoregulation.

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Review

Eur Respir J

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. 2022 Aug 11;2201217.

doi: 10.1183/13993003.01217-2022. Online ahead of print.

Living without eosinophils: evidence from mouse and man

David J Jackson[1,2], Ian D Pavord[3]

Affiliations expand

- PMID: 35953100

- DOI: 10.1183/13993003.01217-2022

Abstract

The enduring view of eosinophils, as immune effector cells whose primary function is host defence against infection by helminths and other microbial pathogens, sets the stage for a fundamental question regarding the safety of therapeutic eosinophil depletion. If eosinophils are significantly reduced or altogether depleted in an effort to alleviate the negative effects of tissue eosinophilia and eosinophilic inflammation in conditions such as asthma, chronic obstructive pulmonary disease, chronic rhinosinusitis with nasal polyps, eosinophilic granulomatosis with polyangiitis and hypereosinophilic syndrome, would these patients become susceptible to infection or another illness? Development of mouse models in whom the eosinophil lineage has been ablated, observations in patients naturally lacking eosinophils and data from studies of eosinophil-depleting medical therapies indicate that the absence of eosinophils is not detrimental to health. The evidence available to date, as presented in this review, supports the conclusion that even if certain homeostatic roles for the eosinophil may be demonstrable in controlled animal models and human *in vitro* settings, the evolution of the human species appears to have provided sufficient immune redundancy such that one may be hale and hearty without eosinophils.

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Review

Allergy

. 2022 Aug 11.

doi: 10.1111/all.15473. Online ahead of print.

Biologics and Airway Remodeling in Severe Asthma

Gilda Varricchi[1 2 3 4], Sebastian Ferri[5], Jack Pepys[6], Remo Poto[1 3 4], Giuseppe Spadaro[1 3 4], Nappi Emanuele[5 6], Giovanni Paoletti[5 6], J Christian Virchow[7], Enrico Heffler[5 6], Walter G Canonica[5 6]

Affiliations expand

- PMID: 35950646

- DOI: 10.1111/all.15473

Abstract

Asthma is a chronic inflammatory airway disease resulting in airflow obstruction, which in part can become irreversible to conventional therapies, defining the concept of airway remodeling. The introduction of biologics in severe asthma has led in some patients to the complete normalization of previously considered irreversible airflow obstruction. This highlights the need to distinguish a "fixed" airflow obstruction due to structural changes unresponsive to current therapies, from a "reversible" one as demonstrated by lung function normalization during biological therapies not previously obtained even with high-dose systemic glucocorticoids. The mechanisms by which exposure to environmental factors initiates the inflammatory responses that trigger airway remodeling are still incompletely understood. Alarmins represent epithelial-derived cytokines that initiate immunologic events leading to inflammatory airway remodeling. Biological therapies can

improve airflow obstruction by addressing these airway inflammatory changes. In addition, biologics might prevent and possibly even revert "fixed" remodeling due to structural changes. Hence, it appears clinically important to separate the therapeutic effects (early and late) of biologics as a new paradigm to evaluate the effects of these drugs and future treatments on airway remodeling in severe asthma.

Keywords: airway remodeling; biologics; biomarkers; immunotherapies; severe asthma.

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Curr Pediatr Rev

. 2022 Aug 10.

doi: 10.2174/1573396318666220810161945. Online ahead of print.

Respiratory syncytial virus is the most common causatives of viral bronchiolitis in young children: An updated Review

Kam Lun Hon[1], Alexander K C Leung[2], Alex H C Wong[3], Amrita Dudi[1], Karen Ka Yan Leung[1]

Affiliations expand

- PMID: 35950255

- DOI: 10.2174/1573396318666220810161945

Abstract

Background: Viral bronchiolitis is a common condition and a leading cause of hospitalization in young children.

Objective: This article provides readers with an update on the evaluation, diagnosis, and treatment of viral bronchiolitis, primarily due to RSV Methods: A PubMed search was conducted in December 2021 in Clinical Queries using the key terms "acute bronchiolitis" OR "respiratory syncytial virus infection". The search included clinical trials, randomized controlled trials, case control studies, cohort studies, meta-analyses, observational studies, clinical guidelines, case reports, case series, and reviews. The search was restricted to children and English literature. The information retrieved from the above search was used in the compilation of this article.

Results: Respiratory syncytial virus (RSV) bronchiolitis is the most common viral bronchiolitis in young children. Other viruses such as human rhinovirus and coronavirus could be etiological agents. Diagnosis is based on clinical manifestation. Viral testing is useful only for cohort and quarantine purposes. Cochrane evidence-based reviews have been performed on most treatment modalities for RSV and various viral bronchiolitis. Treatment for viral bronchiolitis is mainly symptomatic support. Beta-agonists are frequently used despite the lack of evidence that they reduce hospital admissions or length of stay. Nebulized racemic epinephrine, hypertonic saline and corticosteroids are generally not effective. Passive immunoprophylaxis with a monoclonal antibody against RSV, when given intramuscularly and monthly during winter, is effective in preventing severe RSV bronchiolitis in high-risk children who are born prematurely and in children under 2 years with chronic lung disease or hemodynamically significant congenital heart disease. Vaccines for RSV bronchiolitis are being developed. Children with viral bronchiolitis in early life are at increased risk of developing asthma later in childhood Conclusions: Viral bronchiolitis is common. No current pharmacologic treatment or novel therapy has been proven to improve outcomes comparing to supportive treatment. Viral bronchiolitis in early life predisposes asthma development later in childhood.

Keywords: Bronchiolitis; RSV; corticosteroids; hypertonic saline; nebulized racemic epinephrine; respiratory syncytial virus; viral; wheezing; beta-agonists.

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Review

Eur Respir Rev

. 2022 Aug 10;31(165):220038.

doi: 10.1183/16000617.0038-2022. Print 2022 Sep 30.

Air pollution, metabolites and respiratory health across the life-course

Olena Gruzieva[1,2,3], Ayoung Jeong[4,5,3], Shizhen He[6], Zhebin Yu[6], Jeroen de Bont[6], Maria G M Pinho[7], Ikenna C Eze[4,5], Sara Kress[8], Craig E Wheelock[9,10,11], Annette Peters[12], Jelle Vlaanderen[13], Kees de Hoogh[4,5], Augustin Scalbert[14], Marc Chadeau-Hyam[13,15], Roel C H Vermeulen[13], Ulrike Gehring[13,16], Nicole Probst-Hensch[4,5,16], Erik Melén[17,18,16]

Affiliations expand

- PMID: 35948392

- DOI: 10.1183/16000617.0038-2022

Free article

Abstract

Previous studies have explored the relationships of air pollution and metabolic profiles with lung function. However, the metabolites linking air pollution and lung function and the associated mechanisms have not been reviewed from a life-course perspective. Here, we provide a narrative review summarising recent evidence on the associations of metabolic profiles with air pollution exposure and lung function in children and adults. Twenty-six studies identified through a systematic PubMed search were included with 10 studies analysing air pollution-related metabolic profiles and 16 studies analysing lung function-related metabolic profiles. A wide range of metabolites were associated with short- and long-term exposure, partly overlapping with those linked to lung function in the general population and with respiratory diseases such as asthma and COPD. The existing studies show that metabolomics offers the potential to identify biomarkers linked to both environmental exposures and respiratory outcomes, but many studies suffer from small sample sizes, cross-sectional designs, a preponderance on adult lung function, heterogeneity in exposure assessment, lack of confounding control and omics integration. The ongoing EXposome Powered tools for healthy living in urbAN Settings (EXPANSE) project aims to address some of these shortcomings by combining biospecimens from large European cohorts and harmonised air pollution exposure and exposome data.

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Conflict of interest statement

Conflict of interest: M.G.M. Pinho reports a Leadership or fiduciary role in other board, society, committee or advocacy group, paid or unpaid as an Associate editor at the journal Public Health Nutrition. C.E. Wheelock has received support for the present manuscript from Swedish Heart Lung Foundation. M. Chadeau-Hyam has shares in the OSMOSE consulting company; work performed in the company has no link with the present work. N. Probst-Hensch reports salary support from Swiss TPH Core Funds. The remaining authors have nothing to disclose.

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Published Erratum

Exp Mol Med

. 2022 Aug 9.

doi: 10.1038/s12276-022-00826-9. Online ahead of print.

Author Correction: S100A9 in adult asthmatic patients: a biomarker for neutrophilic asthma

Quang Luu Quoc[1,2], Youngwoo Choi[1], Tra Cao Thi Bich[1,2], Eun-Mi Yang[1], Yoo Seob Shin[1,2], Hae-Sim Park[3,4]

Affiliations expand

- PMID: 35945450

- DOI: 10.1038/s12276-022-00826-9

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Erratum for

- S100A9 in adult asthmatic patients: a biomarker for neutrophilic asthma.
  Quoc QL, Choi Y, Thi Bich TC, Yang EM, Shin YS, Park HS.Exp Mol Med. 2021 Jul;53(7):1170-1179. doi: 10.1038/s12276-021-00652-5. Epub 2021 Jul 20.PMID: 34285336 **Free PMC article.**

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Am J Respir Crit Care Med

. 2022 Aug 9.

doi: 10.1164/rccm.202109-2082LE. Online ahead of print.

MMP-12 Supports Pulmonary B Cell Follicle Formation and Local Antibody Responses During Asthma

Chloe J Pyle[1], Dhiren F Patel[1], Teresa Peiró[1,2], Régis Joulia[1], Aleksander M Grabiec[3], Tracy Hussell[3], Gael Tavernier[4,5], Angela Simpson[6], James Pease[7], James A Harker[8], Clare M Lloyd[9], Robert J Snelgrove[10]

Affiliations expand

- PMID: 35944138

- DOI: 10.1164/rccm.202109-2082LE

No abstract available

Keywords: Asthma; MMP-12; Protease.

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J Asthma

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. 2022 Aug 9;1-13.

doi: 10.1080/02770903.2022.2111686. Online ahead of print.

Initial emergency department vital signs may predict PICU admission in pediatric patients presenting with asthma exacerbation

Michael S Freedman[1,2], Erick Forno[3,4]

Affiliations expand

- PMID: 35943201

- DOI: 10.1080/02770903.2022.2111686

Abstract

Objective: Severe asthma exacerbations account for a large share of asthma morbidity, mortality, and costs. Here, we aim to identify early predictive factors associated with pediatric intensive care unit (PICU) admission.

Methods: We performed a retrospective observational study of 5,185 emergency department (ED) encounters at a large children's hospital, including 86 (1.7%) resulting in PICU admission between 10/1/2015 and 8/7/2018 with ICD9/ICD10 codes for "asthma," "bronchospasm," or "wheezing". Vital signs and demographic information were obtained from EHR data and analyzed for each encounter. Predictive factors were identified using adjusted regression models, and our primary outcome was PICU admission.

Results: Higher mean heart rates (HR) and respiratory rates (RR) and lower SpO2 within the first hour of ED presentation were independently associated with PICU admission. Odds of PICU admission increased 70% for each 10-beats/minute higher HR, 125% for each 10-breaths/minute higher RR, and 34% for each 5% lower SpO2. A binary predictive index using 1-hour vitals yielded OR 13.4 (95%CI 8.1-22.1) for PICU admission, area under the receiver operator characteristic curve (AUROC) 0.84 and overall accuracy of 80.1%. Results were largely unchanged (AUROC 0.84-0.88) after adjusting for surrogates of asthma severity and initial ED management. In combination with a secondary standardized clinical asthma distress score, positive predictive value increased by seven-fold (6.1% to 46%).

Conclusions: A predictive index using HR, RR and SpO2 within the first hour of ED presentation accurately predicted PICU admission in this cohort. Automated vital signs trend analysis may help identify vulnerable patients quickly upon presentation.

Keywords: asthma; emergency department; hospital; hospitalization; intensive care units; length of stay; pediatric; vital signs.

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Allergy Asthma Clin Immunol

. 2022 Aug 7;18(1):72.

doi: 10.1186/s13223-022-00713-y.

Optimizing pediatric asthma education using virtual platforms during the COVID-19 pandemic

Dhenuka Radhakrishnan[1 2 3 4], Andrea Higginson[5], Madhura Thipse[6], Marc Tessier[5], Arun Radhakrishnan[7]

Affiliations expand

- PMID: 35934694

- PMCID: PMC9358084

- DOI: 10.1186/s13223-022-00713-y

Free PMC article

Abstract

Background: We compared patient and caregiver knowledge and confidence for managing asthma, and participant experiences when comprehensive asthma education was delivered in person versus in the virtual setting.

Methods: We performed a multi-methods study using structured surveys and qualitative interviews to solicit feedback from patients and caregivers following participation in a comprehensive asthma education session between April 2018 and October 2021. We compared participant knowledge and confidence for managing asthma as well as user experience when the education was attended in-person or virtually. Quantitative responses were summarized descriptively, and qualitative feedback was analyzed for major themes.

Results: Of 100 caregivers/patients who completed post education satisfaction surveys and interviews, 52 attended in person and 48 virtually, with the mean age of patients being 6.7 years (range: 1.2-17.0). Participant reported gains in knowledge and confidence for asthma management were not different between groups and 65.2% preferred attending virtual asthma education. The majority of participants described virtual education as a safer modality that was more convenient and accessible.

Conclusions: We demonstrated the successful implementation of a novel, virtual asthma education program for patients and caregivers of children with asthma. Both virtual and in-person delivered asthma education were equally effective for improving perceived knowledge and confidence for asthma self-management and virtual education was considered safer, more convenient and accessible. Virtual asthma education offers an attractive and effective option for improving the reach of quality asthma education programs and may allow more children/patients to benefit.

Keywords: Asthma; Education; Pandemic; Pediatrics; Telemedicine.

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Conflict of interest statement

The authors declare that they have no competing interests.

- 16 references
- 2 figures

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Pediatr Pulmonol

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. 2022 Aug 7.

doi: 10.1002/ppul.26103. Online ahead of print.

CFTR gene variants, air pollution, and childhood asthma in a California Medicaid population

Ruwan Thilakaratne[1,2], Steve Graham[3], John Moua[4], Caitlin G Jones[1], Caroline Collins[1], Jennifer Mann[1], Stanley Sciortino[3], Jacklyn Wong[1], Martin Kharrazi[1]

Affiliations expand

- PMID: 35933722

- DOI: 10.1002/ppul.26103

Abstract

Carriers of the cystic fibrosis transmembrane conductance regulator (CFTR) gene ("carriers") have been found to have an increased risk of persistent asthma. However, it is unclear at what level of CFTR function this risk exists and whether it is modified by asthmogens, such as air pollution. We conducted a retrospective cohort study of children born in California between July 2007 and December 2013, linking CFTR genotype data from the California newborn screening program to Medicaid claims records through March 17, 2020 to identify asthma cases, and to air pollution data from CalEnviroScreen 3.0 to identify levels of particulate matter with diameter 2.5 microns or smaller ($PM_{2.5}$). Log-binomial regression models for asthma risk were fitted, adjusting for race/ethnicity and sex. Compared to population controls, carriers had higher risk of asthma (adjusted risk ratio (aRR)=1.29, 95% confidence interval (CI): 0.98, 1.69; $p<0.1$). Other non-CF-causing variants on the second allele did not appear to further increase risk. Genotypes with the greatest asthma risk were F508del with an intron 10 T7 or (TG)11T5 in trans (aRR=1.52, 95% CI: 1.10, 2.12). This association was higher among children living in areas at or above (aRR=1.80) versus below (aRR=1.37) the current national air quality standard for $PM_{2.5}$, though this difference was not statistically significant ($p_{interaction}$ >0.2). These results suggest carriers with CFTR functional levels between 25% and 45% of wildtype are at increased risk of asthma. Knowledge of CFTR genotype in asthmatics may be important to open new CFTR-related treatment options for these patients. This article is protected by copyright. All rights reserved.

Keywords: child health; cystic fibrosis transmembrane conductance regulator; gene-environment interaction; neonatal screening.

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J Asthma

. 2022 Aug 11;1-9.

doi: 10.1080/02770903.2022.2103427. Online ahead of print.

Severe non-atopic asthma: omalizumab can reduce severe asthma exacerbations

Lise Melscoet[1], Naji Khayath[1,2], Nicolas Migueres[1], Marc-André Goltzene[3], Nicolas Meyer[3], Frédéric de Blay[1,2]

Affiliations expand

- PMID: 35862624

- DOI: 10.1080/02770903.2022.2103427

Abstract

Introduction: Humanized monoclonal anti-IgE antibody (omalizumab) has demonstrated efficacy in severe atopic asthma. However, few studies have assessed its efficacy in non-atopic and even less in T2-low severe asthma. The objective was to determinate the omalizumab response according to atopic status.

Methods: This retrospective, real-world study was performed in the Chest Diseases Department of Strasbourg University Hospital from January 1, 2006, to June 30, 2017. The response to omalizumab was assessed in 139 patients 4, 6, and 12 months after treatment and compared to data collected prior to omalizumab initiation.

Results: Forty-four patients (31.7%) had severe non-atopic asthma and 95 (68.3%) had a severe atopic asthma. In the non-atopic group, omalizumab significantly reduced the severe exacerbation rate by 44% (95% CI 18-64%, $p < 0.05$), 43% (CI 95% 20-60%, $p < 0.05$), and 54% (CI 95% 36-67%, $p < 0.05$), at 4, 6 and 12 months, respectively. A trend toward improvement in FEV1, asthma control and oral corticosteroid use was also observed. These results were not significantly different from those obtained in atopic asthmatics except a more effective oral corticosteroid sparing in atopic group ($p < 0.05$). Similar reduction of severe exacerbation rates were observed in T2-low asthma subgroup (non-atopic, non-eosinophilic).

Conclusion: Omalizumab was effective in severe asthma, regardless of atopic status.

Keywords: Severe asthma; T2 inflammation; atopy; eosinophil; omalizumab.

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JAMA

. 2022 Aug 9;328(6):575-576.

doi: 10.1001/jama.2022.12258.

Therapy for Mild to Moderate Asthma

Sarah E Gray[1], Adam S Cifu[1], Valerie G Press[1]

Affiliations expand

- PMID: 35862052

- DOI: 10.1001/jama.2022.12258

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Review

Toxicol Appl Pharmacol

. 2022 Aug 15;449:116070.

doi: 10.1016/j.taap.2022.116070. Epub 2022 May 23.

Inflammation resolution in environmental pulmonary health and morbidity

Jacqui M Marzec[1], Srikanth S Nadadur[2]

Affiliations expand

- PMID: 35618031

- DOI: 10.1016/j.taap.2022.116070

Abstract

Inflammation and resolution are dynamic processes comprised of inflammatory activation and neutrophil influx, followed by mediator catabolism and efferocytosis. These critical pathways ensure a return to homeostasis and promote repair. Over the past decade research has shown that diverse mediators play a role in the active process of resolution. Specialized pro-resolving mediators (SPMs), biosynthesized from fatty acids, are released during inflammation to facilitate resolution and are deficient in a variety of lung disorders. Failed resolution results in remodeling and cellular deposition through pro-fibrotic myofibroblast expansion that irreversibly narrows the airways and worsens lung function. Recent studies indicate environmental exposures may perturb and deregulate critical resolution pathways. Environmental xenobiotics induce lung inflammation and generate reactive metabolites that promote oxidative stress, injuring the respiratory mucosa and impairing gas-exchange. This warrants recognition of xenobiotic associated molecular patterns (XAMPs) as new signals in the field of inflammation biology, as many environmental chemicals generate free radicals capable of initiating the inflammatory response. Recent studies suggest that unresolved, persistent inflammation impacts both resolution pathways and endogenous regulatory mediators, compromising lung function, which over time can progress to chronic lung disease. Chronic ozone (O_3) exposure overwhelms successful resolution, and in susceptible individuals promotes asthma onset. The industrial contaminant cadmium (Cd) bioaccumulates in the lung to impair resolution, and recurrent inflammation can result in chronic obstructive pulmonary disease (COPD). Persistent particulate matter (PM) exposure increases systemic cardiopulmonary inflammation, which reduces lung function and can exacerbate asthma, COPD, and idiopathic pulmonary fibrosis (IPF). While recurrent inflammation underlies environmentally induced pulmonary morbidity and may drive the disease process, our understanding of inflammation resolution in this context is limited. This review aims to explore inflammation resolution biology and its role in chronic environmental lung disease(s).

Keywords: ALI; Asthma; COPD; Clearance; Environment; Gasotransmitters; IPF; Inflammation; Oxidative stress; Pollutants; Resolution; SPMs.

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J Infect Dis

. 2022 Aug 12;226(Supplement_1):S55-S62.

doi: 10.1093/infdis/jiac141.

A Retrospective Cohort Study on Infant Respiratory Tract Infection Hospitalizations and Recurrent Wheeze and Asthma Risk: Impact of Respiratory Syncytial Virus

Maarten van Wijhe[1,2], Caroline Klint Johannesen[1,3], Lone Simonsen[2], Inger Merete Jørgensen[4], RESCEU Investigators; Thea K Fischer[1,3,5]

Collaborators, Affiliations expand

- PMID: 35426942

- DOI: 10.1093/infdis/jiac141

Abstract

Background: Infant respiratory syncytial virus infection (RSV) has been associated with asthma later in life. We explored the risk of recurrent wheeze or asthma in children with infant RSV-associated hospitalization compared to other respiratory infections.

Methods: We performed a retrospective cohort study using Danish national hospital discharge registers. Infants younger than 6 months, born between January 1995 and October 2018, and with a RSV hospital admission were compared to infants hospitalized for injuries, non-RSV acute upper respiratory tract infection (AURTI), pneumonia and other respiratory pathogens, nonpathogen-coded lower respiratory tract infections (LRTI), pertussis, or nonspecific respiratory infections. Infants were followed until recurrent wheeze or asthma diagnosis, death, migration, age 10 years, or study end. We estimated cumulative incidence rate ratios (CIRR) and hazard ratios (HR) adjusted for sex, age at inclusion, hospital length of stay (LOS), maternal smoking, 5-minute APGAR score (APGAR5), prematurity, and congenital risk factors (CRF).

Results: We included 68 130 infants, of whom 20 920 (30.7%) had RSV hospitalization. The cumulative incidence rate of recurrent wheeze or asthma was 16.6 per 1000 person-years after RSV hospitalization, higher than after injury (CIRR, 2.69; 95% confidence interval [CI], 2.48-2.92), AURTI (CIRR, 1.48; 95% CI, 1.34-1.58), or pertussis (CIRR, 2.32; 95% CI, 1.85-2.91), similar to pneumonia and other respiratory pathogens (CIRR, 1.15; 95% CI, .99-1.34) and LRTI (CIRR, 0.79; 95% CI, .60-1.04), but lower than nonspecific respiratory infections (CIRR, 0.79; 95% CI, .73-.87). Adjusted HRs for recurrent wheeze or asthma after RSV hospitalization compared to injuries decreased from 2.37 (95% CI, 2.08-2.70) for 0 to <1 year to 1.23 (95% CI, .88-1.73) for 6 to <10 years for term-born children, and from 1.48 (95% CI, 1.09-2.00) to 0.60 (95% CI, .25-1.43) for preterm-born children. Sex, maternal smoking, LOS, CRF, and APGAR5 were independent risk factors.

Conclusions: Infant RSV hospitalization is associated with recurrent wheeze and asthma hospitalization, predominantly at preschool age. If causal, RSV prophylaxis, including vaccines, may significantly reduce disease burden of wheeze and asthma.

Keywords: RSV; asthma; hospitalizations; recurrent wheeze; register study; respiratory syncytial virus; retrospective cohort study.

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Conflict of interest statement

Potential conflicts of interest. All authors: No reported conflicts. All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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J Infect Dis

. 2022 Aug 12;226(Supplement_1):S38-S44.

doi: 10.1093/infdis/jiab308.

Time-Varying Association Between Severe Respiratory Syncytial Virus Infections and Subsequent Severe Asthma and Wheeze and Influences of Age at the Infection

Xin Wang[1,2], You Li[1,2], Harish Nair[1], Harry Campbell[1], RESCEU Investigators

Collaborators, Affiliations expand

- PMID: 34522963

- DOI: 10.1093/infdis/jiab308

Abstract

Background: Early-life severe respiratory syncytial virus (RSV) infection has been associated with subsequent risk of asthma and recurrent wheeze. However, changes in the association over time and the interaction effect of the age at first RSV infection are less

well understood. We aimed to assess the time-varying association between RSV and subsequent asthma and wheeze admission and explore how the association was affected by the age at RSV infection.

Methods: We retrospectively followed up a cohort of 23 365 children for a median of 6.9 years using Scottish health databases. Children who were born between 2001 and 2013 and had RSV-associated respiratory tract infection (RTI) admissions under 2 years were in the exposed group; those with unintentional accident admissions under 2 years comprised the control group. The Cox proportional-hazards model was used to report adjusted hazard ratios (HRs) of RSV admissions on subsequent asthma and wheeze admissions. We did subgroup analyses by follow-up years. We also explored how this association was affected by the age at first RSV admission.

Results: The association was strongest in the first 2 years of follow-up and decreased over time. The association persisted for 6 years in children whose first RSV-RTI admission occurred at 6-23 months of age, with an adjusted HR of 3.9 (95% confidence interval [CI], 3.1-4.9) for the first 2 years, 2.3 (95% CI, 1.6-3.2) for 2 to <4 years, and 1.9 (95% CI, 1.2-2.9) for 4 to <6 years of follow-up. In contrast, the association was only significant for the first 2 years after first RSV-RTI admissions occurring at 0-5 months.

Conclusions: We found a more persistent association for subsequent asthma and wheeze in children whose first severe RSV infection occurred at 6-23 months compared to those whose first severe RSV infection occurred at 0-6 months. This provides new evidence for further assessment of the association and RSV intervention programs.

Keywords: age at first RSV infection; asthma; severe RSV infections; time since RSV infection; wheeze.

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Conflict of interest statement

Potential conflicts of interest. H. C. reports grants from the European Union Innovative Medicines Initiative; grants and personal fees from the World Health Organization; and grants from the Bill & Melinda Gates Foundation during the conduct of the study. H. N. reports grants from World Health Organization, grants and personal fees from Sanofi Pasteur, personal fees from Janssen, personal fees from the Bill & Melinda Gates Foundation, personal fees from Reviral, personal fees from AbbVie, and grants from the National Institute of Health Research outside the submitted work. Y. L. reports grants from the World Health Organization; and grants from Wellcome Trust outside the submitted work. All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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RHINITIS

J Allergy Clin Immunol Pract

. 2022 Aug 12;S2213-2198(22)00809-1.

doi: 10.1016/j.jaip.2022.08.007. Online ahead of print.

Differences between early and late-onset asthma: role of comorbidities on symptom control

Martina Turrin[1], Michele Rizzo[1], Matteo Bonato[1], Erica Bazzan[1], Manuel G Cosio[2], Umberto Semenzato[1], Marina Saetta[3], Simonetta Baraldo[1]

Affiliations expand

- PMID: 35970446

- DOI: 10.1016/j.jaip.2022.08.007

Abstract

Background: Asthma can present in early childhood or de novo in adulthood. Our understanding of the burden of comorbidities in adult asthmatic patients stratified by age of onset is incomplete.

Objectives: To evaluate how different comorbidities would affect symptom control in two distinct groups of patients with early and late onset asthma (EOA and LOA). To explore whether reported comorbidities associate with lung function and inflammatory parameters.

Methods: We conducted a cross-sectional study of 175 adult asthmatics (57.5±17.1 years) recruited at our University Asthma Clinic. We defined EOA as asthma onset <12 years; LOA as onset >40 years. The primary outcome was symptom control and the main comorbidities evaluated were rhinitis, GERD, obesity, cardiovascular and bronchiectasis. Multivariable regression analysis was used to identify potential predictors of poor control in EOA and LOA.

Results: 77/175 (44%) subjects had EOA, 98 (56%) had LOA and comorbidities had a differential impact in the two groups. Rhinitis was more frequent in EOA (76 vs 53%;p=0.02) and was associated with uncontrolled asthma (p<0.001), reduced FEV_1/FVC (p=0.01), increased eosinophils (p=0.003) and total IgE (p<0.01). Conversely, in LOA rhinitis was associated with more controlled asthma and higher FEV_1/FVC (both p<0.01). In EOA only IgE levels were directly related to blood eosinophils (r=0.42;p<0.001) and inversely to FEV1/FVC (r=- 0.35;p=0.002). Obesity was present in 20% of patients in both groups, but only in LOA obesity was associated to uncontrolled disease (p=0.009), reduced FEV_1/FVC (p=0.009) and blood neutrophils (p=0.03). In multivariable regression analysis rhinitis in EOA and obesity in LOA were the risk factor most closely associated with poor control; GERD, cardiovascular and bronchiectasis did not affect control.

Conclusions: Early-onset persistent and late-onset asthma are distinct phenotypes, with different underlying inflammatory patterns and different comorbidities which impact on symptom control.

Keywords: Age of onset; asthma control; comorbidities; inflammation; lung function.

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Risk Manag Healthc Policy

. 2022 Aug 8;15:1501-1515.

doi: 10.2147/RMHP.S373085. eCollection 2022.

The Association Between Air Pollutants and Daily Outpatient Visits for Allergic Rhinitis: A Time-Series Analysis Based on Distribution Lag Nonlinear Model in Chongqing, China

Dan Yang[1], Yongjie Yan[1], Kexue Pu[1,2]

Affiliations expand

- PMID: 35966499
- PMCID: PMC9374097
- DOI: 10.2147/RMHP.S373085

Abstract

Background: Allergic rhinitis (AR) is a severe and the most common chronic allergic disease, affecting 10-40% of the world population. The effect of air pollutants on AR has been confirmed in clinical experiments.

Purpose: This study aimed to quantify the association between air pollutants and daily outpatient visits for AR in Chongqing, China.

Methods: Based on the data of AR outpatients in the primary urban area of Chongqing from 2016 to 2017, along with the atmospheric pollutants and meteorological data in the same period, the distributed lag nonlinear model (DLNM) and generalized additive model (GAM) were used to analyze the time-series. We examined the effects of the single and double pollutant models with a maximum lag day of 30 days. Effect estimates were described as relative risk (RR) and 95% confidence intervals (CIs) in daily outpatient visits for AR per 10 μg/m^3 increases in $PM_{2.5}$, PM_{10}, SO_2, NO_2, O_3, and per 1 mg/m^3 increase in CO.

Results: A single pollutant's O_3 level had an immediate positive effect on AR within two days, the relative risks (RR, 95% CI) were 1.066 (1.008-1.127), 1.057 (1.005-1.112) and 1.048 (1.002-1.097). $PM_{2.5}$ had a lag effect within 11-18 days, the max relative risks (RR, 95% CI) were 1.083 (1.010-1.160). Moreover, O_3, $PM_{2.5}$, PM_{10}, SO_2 and NO_2 had significant effects on AR in the two-pollutant model. The RR cumulative effect of $PM_{2.5}$ became more

pronounced as the concentration increased. The cumulative effect of NO_2 was lesser than $PM_{2.5}$.

Conclusion: Air pollutants were associated with the daily outpatient visits for AR, which may have considerable implications for developing tailored health policies and services to prevent AR in Chongqing and even all over the world.

Keywords: air pollution; allergic rhinitis; distributed lag nonlinear model; generalized additive model; time-series analysis.

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Conflict of interest statement

The authors report no conflicts of interest in relation to this work and declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

- 33 references
- 6 figures

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Editorial

J Allergy Clin Immunol

. 2022 Aug 11;S0091-6749(22)01052-1.

doi: 10.1016/j.jaci.2022.08.001. Online ahead of print.

Regulation of the IgE Response by T Follicular Regulatory Cells

Qiang Chen[1], Alexander L Dent[2]

Affiliations expand

- PMID: 35964780

- DOI: 10.1016/j.jaci.2022.08.001

No abstract available

Keywords: IgE; T follicular regulatory cells; allergic airway inflammation; allergic rhinitis; food allergy.

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J Aerosol Med Pulm Drug Deliv

. 2022 Aug 12.

doi: 10.1089/jamp.2022.0025. Online ahead of print.

Sodium Pyruvate Nasal Spray Reduces the Severity of Nasal Inflammation and Congestion in Patients with Allergic Rhinitis

Alain Martin[1], Christopher Lupfer[2], Ronald Amen[1]

Affiliations expand

- PMID: 35960504

- DOI: 10.1089/jamp.2022.0025

Abstract

Background: As an anti-inflammatory and antioxidant, sodium pyruvate significantly reduces inflammatory cytokines and oxygen radicals such as interleukin (IL) IL-6, IL-8, Monocyte Chemoattractant Protein-1, and hydrogen peroxide. Thus, sodium pyruvate holds promise as a treatment for many respiratory diseases, including allergic rhinitis (AR). Novel treatments for AR are needed as current medications, including steroids, often fail to treat severe symptoms. ***Methods:*** The data from five human clinical studies were analyzed to determine the effect of 20 mM sodium pyruvate nasal spray (N115) in patients with AR. Nasal inflammation scores were compared to a placebo control or a no-treatment baseline control. Three studies were open-labeled and two were appropriately blinded to both patients and clinicians using computer randomization of subjects. ***Results:*** The intranasal administration of sodium pyruvate significantly improved nasal inflammation scores in all five clinical trials of patients with AR ($p < 0.0001$ in all trials). ***Conclusions:*** These results give credence to the overall ability of sodium pyruvate, administered by nasal spray, to treat inflammation of the nasal airways.

Keywords: allergic rhinitis; anti-inflammatory; inhaled pyruvate.

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Review

Clin Mol Allergy

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. 2022 Aug 10;20(1):9.

doi: 10.1186/s12948-022-00176-x.

Current treatment strategies for seasonal allergic rhinitis: where are we heading?

Erminia Ridolo[1], Cristoforo Incorvaia[2], Francesco Pucciarini[2], Elena Makri[3], Giovanni Paoletti[4,5], Giorgio Walter Canonica[4,5]

Affiliations expand

- PMID: 35948975
- PMCID: PMC9367100
- DOI: 10.1186/s12948-022-00176-x

Abstract

Introduction: Allergic rhinitis (AR) is very commonly caused by pollens. The symptoms of AR consist of sneezing, nasal congestion, rhinorrhea, nasal itching and airflow obstruction. The diagnosis has long been based on clinical history, skin prick tests and in vitro measurement of specific IgE, but the innovative approach of precision medicine has made diagnostic tools of much greater accuracy available.

Areas covered: This review covers the advances in the treatment of seasonal AR concerning the drugs to be used according to the grade of disease and the characteristics of the patients, and the role of allergen immunotherapy (AIT), which is the only treatment capable of acting, in addition to the symptoms, on the cause of AR and therefore to modify its natural history.

Expert opinion: Drug treatment of AR include a large number of agents, the choice of which depends on the severity of the disease. AIT has high evidence of efficacy demonstrated by meta-analyses, and further improvement is currently apparent, as for diagnosis, applying the means of precision medicine. However, when AIT is performed in current practice, without the strict rules of controlled trials, long-term low adherence is a major problem to be solved.

Keywords: Allergen immunotherapy; Personalized medicine; Seasonal allergic rhinitis; Symptomatic drugs; Treatment strategy.

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Conflict of interest statement

The authors declare that they have no competing interests.

- 48 references
- 1 figure

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Review

Clin Exp Allergy

. 2022 Aug 10.

doi: 10.1111/cea.14212. Online ahead of print.

Pharmacology, particle deposition and drug administration techniques of intranasal corticosteroids for treating allergic rhinitis

C Rollema[1], E N van Roon[1,2], J F M van Boven[3,4], P Hagedoorn[5], T Klemmeier[6], J W H Kocks[4,7,8,9], E I Metting[10,11], J N G Oude Elberink[4,12], T T A Peters[13], M R M San Giorgi[14], T W de Vries[15]

Affiliations expand

- PMID: 35947495

- DOI: 10.1111/cea.14212

Abstract

This review presents an overview of the available literature regarding intranasal corticosteroids (INCs) for the treatment of allergic rhinitis (AR). Various treatment options exist for AR including INCs, antihistamines and leukotriene antagonists. INCs are considered to be the most effective therapy for moderate to severe AR, as they are effective against nasal and ocular symptoms and improve quality of life. Their safety has been widely observed. INCs are effective and safe for short-term use. Local adverse events are observed but generally well-tolerated. The occurrence of (serious) systemic adverse events is unlikely but cannot be ruled out. There is a lack of long-term safety data. INC may cause serious eye complications. The risk of INCs on the hypothalamic-pituitary-adrenal axis, on bone mineral density reduction or osteoporosis and on growth in children should be considered during treatment. Pharmacological characteristics of INCs (e.g. the mode of action and pharmacokinetics) are well known and described. We sought to gain insight into whether specific properties affect the efficacy and safety of INCs, including nasal particle deposition, which the administration technique affects. However, advances are lacking regarding the improved understanding of the effect of particle deposition on efficacy and safety and the effect of the administration technique. This review emphasizes the gaps in knowledge regarding this subject. Advances in research and healthcare are necessary to improve care for patients with AR.

Keywords: Administration technique; Allergic rhinitis; Intranasal corticosteroids; Nasal particle deposition.

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Laryngoscope

. 2022 Aug 8.

doi: 10.1002/lary.30337. Online ahead of print.

Is Posterior Nasal Nerve Ablation Effective in Treating Symptoms of Allergic Rhinitis?

Camron Davies[1], Daniel Gorelik[2], Andrew P Lane[3], Masayoshi Takashima[2], Omar G Ahmed[2]

Affiliations expand

- PMID: 35938876

- DOI: 10.1002/lary.30337

No abstract available

- 5 references

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CHRONIC COUGH

Pulm Ther

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. 2022 Aug 15.

doi: 10.1007/s41030-022-00193-w. Online ahead of print.

Treatment with the P2X3-Receptor Antagonist Gefapixant for Acute Cough in Induced Viral Upper Respiratory Tract Infection: A Phase 2a, Randomized, Placebo-Controlled Trial

Jaclyn A Smith[1], Michael M Kitt[2], Alan Bell[3], Nicolas Noulin[3], Anjela Tzontcheva[4], Megan McGratty Seng[4], Susan Lu[4]

Affiliations expand

- PMID: 35969360

- DOI: 10.1007/s41030-022-00193-w

Abstract

Introduction: Available therapies for acute cough, a condition frequently caused by a viral upper respiratory tract infection (URTI), have shown limited evidence of efficacy. Gefapixant, a P2X3-receptor antagonist, has demonstrated efficacy and safety in studies of the treatment of refractory or unexplained chronic cough, but its efficacy for treating acute cough has not been previously studied.

Methods: This was a phase 2a, randomized, double-blind, placebo-controlled, parallel-group, pilot study. Healthy volunteers were randomized 1:1 to receive twice-daily gefapixant 45 mg or placebo and inoculated with human rhinovirus 16 to induce URTI and cough. Participants were observed while quarantined for 7 days after the start of treatment. The primary endpoint was awake cough frequency on day 3, which was objectively measured with a cough-recording device. Secondary endpoints included change from baseline to day 3 in subjective cough severity measures (cough severity visual analog scale, Cough Severity Diary) and cough-specific quality of life (Leicester Cough Questionnaire-acute).

Results: Of the 46 participants who met inclusion criteria [mean (standard deviation, SD) age, 24.6 (6.5) years; females, n = 8], 40 completed the study (gefapixant, n = 21; placebo,

n = 19). There was no significant difference in awake cough frequency on day 3 between the gefapixant and placebo groups [least squares means, 2.4 versus 2.7 coughs per hour, respectively; mean difference (95% confidence interval, CI), -0.3 (-2.3, 1.7); P = 0.75]. There were no significant between-group differences for any of the secondary endpoints. Peak cough frequency was low and occurred later in the study than expected (days 4-5). The safety profile was consistent with that of previous studies of gefapixant.

Conclusion: Compared with placebo, gefapixant did not reduce the frequency or severity of acute cough secondary to induced URTI. Induced viral URTI produced mild symptoms, including lower cough frequency than observed in previous studies of patients selected for acute cough associated with naturally occurring URTI.

Trial registration: ClinicalTrials.gov, NCT03569033; EudraCT, 2017-000472-28; protocol number, MK-7264-013.

Keywords: Acute cough; Antitussives; Common cold; Cough frequency; P2X3-receptor antagonists; URTI.

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- 33 references

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Lancet Reg Health Eur

. 2022 Oct;21:100473.

doi: 10.1016/j.lanepe.2022.100473. Epub 2022 Aug 8.

Incidence of community acquired lower respiratory tract disease in Bristol, UK during the COVID-19 pandemic: A prospective cohort study

Catherine Hyams[1,2], Robert Challen[3], Elizabeth Begier[4], Jo Southern[5], Jade King[6], Anna Morley[2], Zsuzsa Szasz-Benczur[1], Maria Garcia Gonzalez[1], Jane Kinney[1], James Campling[5], Sharon Gray[7], Jennifer Oliver[5], Robin Hubler[7], Srinivas Valluri[7], Andrew Vyse[5], John M McLaughlin[7], Gillian Ellsbury[5], Nick A Maskell[2], Bradford D Gessner[7], Leon Danon[3], Adam Finn[1], Avon CAP Research Group

Collaborators, Affiliations expand

- PMID: 35965672

- PMCID: PMC9359590

- DOI: 10.1016/j.lanepe.2022.100473

Abstract

Background: The emergence of COVID-19 and public health measures implemented to reduce SARS-CoV-2 infections have both affected acute lower respiratory tract disease (aLRTD) epidemiology and incidence trends. The severity of COVID-19 and non-SARS-CoV-2 aLRTD during this period have not been compared in detail.

Methods: We conducted a prospective cohort study of adults age ≥18 years admitted to either of two acute care hospitals in Bristol, UK, from August 2020 to November 2021. Patients were included if they presented with signs or symptoms of aLRTD (e.g., cough, pleurisy), or a clinical or radiological aLRTD diagnosis.

Findings: 12,557 adult aLRTD hospitalisations occurred: 10,087 were associated with infection (pneumonia or non-pneumonic lower respiratory tract infection [NP-LRTI]), 2161 with no infective cause, with 306 providing a minimal surveillance dataset. Confirmed SARS-CoV-2 infection accounted for 32% (3178/10,087) of respiratory infections. Annual incidences of overall, COVID-19, and non- SARS-CoV-2 pneumonia were 714.1, 264.2, and 449.9, and NP-LRTI were 346.2, 43.8, and 302.4 per 100,000 adults, respectively. Weekly incidence trends in COVID-19 aLRTD showed large surges (median 6.5 [IQR 0.7-10.2]

admissions per 100,000 adults per week), while other infective aLRTD events were more stable (median 14.3 [IQR 12.8-16.4] admissions per 100,000 adults per week) as were non-infective aLRTD events (median 4.4 [IQR 3.5-5.5] admissions per 100,000 adults per week).

Interpretation: While COVID-19 disease was a large component of total aLRTD during this pandemic period, non- SARS-CoV-2 infection still caused the majority of respiratory infection hospitalisations. COVID-19 disease showed significant temporal fluctuations in frequency, which were less apparent in non-SARS-CoV-2 infection. Despite public health interventions to reduce respiratory infection, disease incidence remains high.

Funding: AvonCAP is an investigator-led project funded under a collaborative agreement by Pfizer.

Keywords: CAP, community acquired pneumonia; COPD, chronic obstructive pulmonary disease; COVID-19; COVID-19, Coronavirus disease 2019; CRDE, chronic respiratory disease exacerbation; Cardiac failure; HF, heart failure; Lower respiratory tract infection; NP-LRTI, non-pneumonic lower respiratory tract infection; Pneumonia; SARS-CoV-2; aLRTD, acute lower respiratory tract disease.

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Conflict of interest statement

CH is Principal Investigator of the Avon CAP study which is an investigator-led University of Bristol study funded by Pfizer and has previously received support from the NIHR in an Academic Clinical Fellowship. JO is a Co-Investigator on the Avon CAP Study. LD is further supported by UKRI through the JUNIPER consortium (grant number MR/V038613/1), MRC (grant number MC/PC/19067), EPSRC (EP/V051555/1 and The Alan Turing Institute, grant EP/N510129/1). AF is a member of the Joint Committee on Vaccination and Immunization (JCVI) and chair of the World Health Organization European Technical Advisory Group of Experts on Immunization (ETAGE) committee. In addition to receiving funding from Pfizer as Chief Investigator of this study, he leads another project investigating transmission of respiratory bacteria in families jointly funded by Pfizer and the Gates Foundation and is an investigator in trials of COVID19 vaccines including ChAdOx1nCOV-19, Janssen and Valneva vaccines. EB, JS, JC, SG, RH, SV, AV, JM, GE, and BG are employees of Pfizer and own Pfizer stock. The other authors have no relevant conflicts of interest to declare. The AvonCAP study is a University of Bristol sponsored study which is investigator-led, and funded under a collaborative agreement by Pfizer Inc.

- 31 references
- 3 figures

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Respirol Case Rep

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. 2022 Jul 4;10(8):e01000.

doi: 10.1002/rcr2.1000. eCollection 2022 Aug.

An endotracheal tumour in a 59-year-old man with chronic cough and episodic haemoptysis

Mohammed Amine Bouanzoul[1]

Affiliations expand

- PMID: 35814193

- PMCID: PMC9251850

- DOI: 10.1002/rcr2.1000

Free PMC article

Abstract

Endotracheal paraganglioma is a rare disease, with less than 20 cases reported globally. We present images of a patient with endotracheal paraganglioma unveiled following chronic cough and episodic haemoptysis.

Keywords: chromaffin; paraganglioma; trachea; vascularization.

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Conflict of interest statement

None declared.

- Cited by 1 article
- 2 references
- 1 figure

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Randomized Controlled Trial

Clin Pharmacokinet

. 2022 Aug;61(8):1143-1156.

doi: 10.1007/s40262-022-01126-1. Epub 2022 May 28.

Safety, Pharmacodynamics, and Pharmacokinetics of P2X3 Receptor Antagonist Eliapixant (BAY 1817080) in Healthy Subjects: Double-Blind Randomized Study

Christian Friedrich[1], Klaus Francke[2], Isabella Gashaw[2,3], Christian Scheerans[2], Stefan Klein[2], Lueder Fels[2], Jaclyn A Smith[4], Thomas Hummel[5], Alyn Morice[6]

Affiliations expand

- PMID: 35624408

- PMCID: PMC9349145

- DOI: 10.1007/s40262-022-01126-1

Free PMC article

Abstract

Background and objective: There is no licensed treatment for refractory chronic cough; off-label therapies have limited efficacy and can produce adverse effects. Excessive adenosine triphosphate signaling via P2X3 receptors is implicated in refractory chronic cough, and selective P2X3 receptor antagonists such as eliapixant (BAY 1817080) are under investigation. The objective of the study was to investigate the safety and tolerability of ascending repeated oral doses of eliapixant in healthy volunteers.

Methods: We conducted a repeated-dose, double-blind, randomized, placebo-controlled study in 47 healthy male individuals. Subjects received repeated twice-daily ascending oral doses of eliapixant (10, 50, 200, and 750 mg) or placebo for 2 weeks. The primary outcome was frequency and severity of adverse events. Other outcomes included pharmacokinetics and evaluation of taste disturbances, which have occurred with the less selective P2X3 receptor antagonist gefapixant.

Results: Peak plasma concentrations of eliapixant were reached 3-4 h after administration of the first and subsequent doses. With multiple dosing, steady-state plasma concentrations were reached after ~ 6 days, and plasma concentrations predicted to achieve ≥ 80% P2X3 receptor occupancy (the level required for efficacy) were reached at 200 and 750 mg. Increases in plasma concentrations with increasing doses were less than dose proportional. After multiple dosing, mean plasma concentrations of eliapixant showed low peak-trough fluctuations and were similar for 200- and 750-mg doses. Eliapixant was well tolerated with a low incidence of taste-related adverse events.

Conclusions: Eliapixant (200 and 750 mg) produced plasma concentrations that cover the predicted therapeutic threshold over 24 h, with good safety and tolerability. These results enabled eliapixant to progress to clinical trials in patients with refractory chronic cough.

Clinical trial registration: Clinicaltrials.gov: NCT03310645 (initial registration: 16 October, 2017).

Plain language summary

There are few effective treatments for patients with a long-term (chronic) cough. It is thought that chronic cough is caused by nerves becoming oversensitive, wrongly causing a cough when there is no need. We tested a new drug called eliapixant in 47 healthy men. Eliapixant reduces the excessive nerve signaling responsible for chronic cough. We looked for side effects of eliapixant and measured how it behaves in the body. In particular we looked for side effects relating to the sense of taste because gefapixant, a similar drug to eliapixant, can affect taste. Participants took one of four eliapixant doses or a placebo twice daily for 2 weeks. The highest levels of eliapixant in the blood were seen 3–4 h after taking the drug, and stable concentrations were seen after about 6 days. At the two highest doses, eliapixant reached concentrations in the body that should be high enough to work in patients with chronic cough. Side effects were generally similar between eliapixant and placebo. Taste-related side effects were mild and went away without needing treatment. The positive results of this study meant that eliapixant could be tested in patients with chronic cough.

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Conflict of interest statement

Christian Friedrich, Klaus Francke, Christian Scheerans, Stefan Klein, and Lueder Fels are employees of Bayer AG, Berlin, Germany. Isabella Gashaw was an employee of Bayer AG, Berlin, Germany, when the study was planned, conducted, and analyzed. Alyn Morice reports grants, personal fees, non-financial support, and other from Bayer AG, and grants, personal fees, non-financial support, and other from Bayer US, during the conduct of the study; personal fees and non-financial support from AstraZeneca, personal fees, non-financial support, and other from Bellus Health, personal fees and non-financial support from Boehringer Ingelheim, personal fees and non-financial support from Chiesi Ltd, grants, personal fees, and non-financial support from GlaxoSmithKline, grants, personal fees, non-financial support, and other from Merck Sharp & Dohme Corp., grants from Menlo Therapeutics, grants, personal fees, and other from NeRRe Therapeutics, grants, personal fees, and non-financial support from Phillips Respironics, grants, personal fees, and non-financial support from Respivant Sciences, Inc., and grants, personal fees, non-financial support, and other from Sanofi, outside the submitted work. Jaclyn A. Smith reports grants and personal fees from Bayer AG during the conduct of the study, personal fees from Algernon Pharmaceuticals, AstraZeneca, and Boehringer Ingelheim, grants and personal fees from Axalbion, Bellus Health, GlaxoSmithKline, Menlo Therapeutics, Merck Sharp & Dohme Corp., NeRRe Therapeutics, Nocion Therapeutics, and Shionogi Inc., outside the submitted work. The VitaloJAK algorithm has been licensed by Manchester University Foundation Trust and the University of Manchester to Vitalograph Ltd. and Vitalograph Ireland (Ltd.). Manchester University Foundation Trust receives royalties, which

may be shared with the clinical division in which Jaclyn A. Smith works. Thomas Hummel reports grants from aspUraclip, Berlin, Germany, Smell and Taste Laboratory, Geneva, Switzerland, Sony, Stuttgart, Germany, and Takasago, Paris, France, and personal fees from Frequency Therapeutics, Farmington, CT, USA and Baiafoods, Madrid, Spain, outside the submitted work.

- Cited by 1 article
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Curr Med Res Opin

. 2022 Aug;38(8):1459-1466.

doi: 10.1080/03007995.2022.2057154. Epub 2022 Apr 15.

Understanding physician's knowledge and perception of chronic cough in Switzerland

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Affiliations expand

- PMID: 35369836

- DOI: 10.1080/03007995.2022.2057154

Abstract

Background: Cough is one of the most common health issues for which medical attention is sought. A chronic cough (CC) is understood as a cough that lasts longer than 8 weeks. CC encompasses two subsets referred to as refractory chronic cough (RCC) and unexplained chronic cough (UCC). This study aims to assess the current understanding and perceptions of a RCC and UCC, from a physician's perspective in Switzerland and how this understanding and practical work leads to the relevant diagnosis and treatment.

Methods: In October 2020, 549 GPs and 338 pulmonologists in Switzerland, received an invite to participate in the online-based quantitative survey. Data collection was carried out through a 25-minute online survey. The questionnaire was based on structured questions, and conducted on a randomized sample of doctors (general practitioners -GPs and pulmonologists) in the German- and French-speaking part of Switzerland.

Results: Overall, 33 pulmonologists and 52 GPs participated in the online survey. Only 39% of GPs, but 73% of pulmonologists, defined chronic cough as a cough lasting 8 weeks or longer. The majority of physicians (72%), especially pulmonologists (88%), perceived a clinical gap regarding the treatment of persistent cough. 74% of the sampled physicians agreed that persistent cough is a high burden of disease for patients. Based on the answers, the annual number of new patients with RCC and UCC in Switzerland is estimated at 9322 patients.

Conclusions: Results of this study have highlighted differences in the terminology used to describe CC (RCC and UCC), in the diagnostic tests used and, in the treatments used between GPs and pulmonologists. These findings suggest the need to align the current language regarding the disease to facilitate a standardized approach for diagnosis and treatment and towards improving patient care and reduce burden of disease for CC (RCC and UCC) patients.

Keywords: Chronic cough; Switzerland; general practitioner; pulmonologists; refractory chronic cough; unexplained chronic cough.

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Minerva Pediatr (Torino)

. 2022 Aug;74(4):491-493.

doi: 10.23736/S2724-5276.22.06807-0. Epub 2022 Feb 10.

Clinical efficacy of sequential therapy with apomycin in children with mycoplasma pneumoniae infection and chronic cough

Lin Zhang[1], Kaiyan Yang[1], Chuan Huang[1], Xinyi Li[1], Zhao Hua[2]

Affiliations expand

- PMID: 35142455

- DOI: 10.23736/S2724-5276.22.06807-0

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Int Forum Allergy Rhinol

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Outcomes of dupilumab treatment versus endoscopic sinus surgery for chronic rhinosinusitis with nasal polyps

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- PMID: 34919344

- DOI: 10.1002/alr.22951

Abstract

Background: Outside of SINUS-24 (A Controlled Clinical Study of Dupilumab in Patients With Bilateral Nasal Polyps) and SINUS-52 (Controlled Clinical Study of Dupilumab in Patients With Nasal Polyps), there are limited data on the efficacy of dupilumab in patients with chronic rhinosinusitis with nasal polyps (CRSwNP). The objective was to compare dupilumab with functional endoscopic sinus surgery (FESS) in patients with CRSwNP by assessing the change in nasal polyp and Sino-Nasal Outcome Test (SNOT-22) scores postintervention.

Methods: A retrospective matched cohort study compared 54 patients who had CRSwNP treated with dupilumab with 54 patients who had CRSwNP treated with FESS; both cohorts were treated with topical corticosteroids. The primary end points were change in nasal polyp score and overall SNOT-22 score. Secondary end points were change in SNOT-22 domain scores and SNOT-22 olfaction score.

Results: Patients who underwent FESS had a greater improvement in nasal polyp score (5.18 ± 2.01) compared with patients treated with dupilumab (4.27 ± 1.98, $p = 0.02$). There was no significant difference in terms of the change in overall SNOT-22 score. Patients treated with dupilumab had greater improvement in the extranasal rhinologic SNOT-22 domain scores (4.87 ± 3.91) compared with patients treated with FESS (2.93 ± 4.32, $p = 0.02$). There was a greater improvement in the SNOT-22 olfaction scores for patients treated with dupilumab (2.35 ± 2.17) compared with patients treated with FESS (1.48 ± 2.24, $p = 0.04$). Patients taking dupilumab were followed on average for 12.20 months and patients treated with FESS were followed for 17.90 months.

Conclusion: Overall, both therapies are effective at reducing symptoms in patients with CRSwNP according to SNOT-22. Patients treated with dupilumab reported improved olfaction and decreased cough, postnasal drainage, and thick nasal drainage as compared with patients treated with FESS, while patients treated with FESS had a greater reduction in polyp burden.

Keywords: biologics; chronic rhinosinusitis; dupilumab; nasal polyps.

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