




ARIA-EAACI statement on asthma and COVID-19 (June 2, 2020)

Jean Bousquet^{1,2,3,4} | Marek Jutel⁵ | Cezmi A. Akdis⁶  | Ludger Klimek⁷ |
 Oliver Pfaar⁸  | Kari C. Nadeau⁹  | Thomas Eiwegger¹⁰  | Anna Bedbrook⁴ |
 Ignacio J. Ansotegui¹¹ | Josep M. Anto^{12,13,14,15} | Claus Bachert^{16,17,18,19}  |
 Eric D. Bateman²⁰ | Kazi S. Bennoor²¹ | Elena Camelia Bergha^{22,23} |
 Karl-Christian Bergmann^{1,2}  | Hubert Blain^{24,25} | Mateo Bonini^{26,27}  |
 Sinthia Bosnic-Anticevich^{28,29} | Louis-Philippe Boulet³⁰  | Luisa Brussino³¹ |
 Roland Buhl³² | Paulo Camargos³³ | Giorgio Walter Canonica³⁴ | Victoria Cardona³⁵  |
 Thomas Casale³⁶  | Sharon Chinthrajah⁹ | Mübeccel Akdis⁶  | Tomas Chivato³⁷ |
 George Christoff³⁸ | Alvaro A. Cruz³⁹  | Wienczyslawa Czarlewski⁴⁰ |
 Stefano Del Giacco⁴¹ | Hui Du⁴² | Yehia El-Gamal⁴³ | Wytske J. Fokkens^{44,45}  |
 Joao A. Fonseca^{46,47,48,49} | Yadong Gao⁵⁰ | Mina Gaga⁵¹ | Bilun Gemicioglu⁵² |
 Maia Gotua⁵³  | Tari Haahtela⁵⁴  | David Halpin⁵⁵ | Eckard Hamelmann⁵⁶  |
 Karin Hoffmann-Sommergruber⁵⁷ | Marc Humbert⁵⁸ | Nataliya Ilina⁵⁹ |
 Juan-Carlos Ivancevich⁶⁰ | Guy Joos⁶¹ | Musa Khaitov⁵⁹ | Bruce Kirenga⁶² |
 Edward F. Knol⁶³ | Fanny W. Ko⁶⁴ | Seppo Koskinen⁶⁵ | Marek L. Kowalski⁶⁶ |
 Helga Kraxner⁶⁷ | Dmitry Kudlay⁵⁹  | Piotr Kuna⁶⁸ | Maciej Kupczyk⁶⁸ |
 Violeta Kvedariene^{69,70} | Amir H. Abdul Latiff⁷¹ | Lan T. Le⁷² | Michael Levin⁷³  |
 Desiree Larenas-Linnemann⁷⁴  | Renaud Louis⁷⁵ | Mohammad R. Masjedi⁷⁶ |
 Erik Melén^{77,78} | Florin Mihaltan⁷⁹ | Branislava Milenkovic⁸⁰ | Yousser Mohammad^{81,82} |
 Mario Morais-Almeida⁸³  | Joaquim Mullol^{84,85} | Leyla Namazova^{86,87} |
 Hugo Neffen^{88,89} | Elisabete Nunes⁹⁰ | Paul O'Byrne^{91,92} | Robyn O'Hehir⁹³ |
 Liam O'Mahony⁹⁴ | Ken Ohta⁹⁵  | Yoshitaka Okamoto⁹⁶ | Gabrielle L. Onorato⁴ |
 Petr Panzner⁹⁷ | Nikos G. Papadopoulos⁹⁸  | Gianni Passalacqua⁹⁹  |
 Vincenzo Patella¹⁰⁰  | Ruby Pawankar¹⁰¹ | Nhat Pham-Thi¹⁰² | Bernard Pigearias¹⁰³ |
 Todor A. Popov¹⁰⁴ | Francesca Puggioni³⁴ | Frederico S. Regateiro^{105,106,107} |
 Giovanni Rolla³¹ | Menachem Rottem^{108,109} | Boleslaw Samolinski¹¹⁰ |
 Joaquin Sastre¹¹¹  | Jurgen Schwarze¹¹² | Aziz Sheikh¹¹³ | Nicola Scichilone¹¹⁴ |
 Manuel Soto-Quiros¹¹⁵ | Manuel Soto-Martinez¹¹⁵ | Milan Sova¹¹⁶ | Stefania Nicola¹¹⁷ |
 Rafael Stelmach¹¹⁸ | Charlotte Suppli-Ulrik¹¹⁹ | Luis Taborda-Barata^{120,121} |

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Teresa To¹²² | **Peter-Valentin Tomazic**¹²³  | **Sanna Toppila-Salmi**⁵⁴  |
Ioanna Tsiligianni^{124,125} | **Omar Usmani**¹²⁶ | **Arunas Valiulis**^{127,128} |
Maria Teresa Ventura¹²⁹ | **Giovanni Viegi**^{130,131} | **Theodor Vontetsianos**¹³² |
De Yun Wang¹³³ | **Sian Williams**¹³⁴ | **Gary W. K. Wong**¹³⁵  | **Arzu Yorgancioglu**¹³⁶ |
Mario Zernotti¹³⁷ | **Mihaela Zidarn**¹³⁸ | **Torsten Zuberbier**^{1,2}  | **Ioana Agache**¹³⁹ 

¹Charité, Universitätsmedizin Berlin, Humboldt-Universität zu Berlin, Berlin, Germany

²Comprehensive Allergy Center, Department of Dermatology and Allergy, Berlin Institute of Health, Berlin, Germany

³University Hospital Montpellier, Montpellier, France

⁴MACVIA-France, Montpellier, France

⁵Department of Clinical Immunology, Wrocław Medical University and ALL-MED Medical Research Institute, Wrocław, Poland

⁶Akdis M. Swiss Institute of Allergy and Asthma Research (SIAF), University of Zurich, Davos, Switzerland

⁷Center for Rhinology and Allergology, Wiesbaden, Germany

⁸Section of Rhinology and Allergy, Department of Otorhinolaryngology, Head and Neck Surgery, University Hospital Marburg, Philipps-Universität Marburg, Marburg, Germany

⁹Stanford University School of Medicine, Sean N. Parker Center for Allergy and Asthma Research, Stanford, CA, USA

¹⁰The Hospital for Sick Children, Department of Paediatrics, Division of Clinical Immunology and Allergy, Food allergy and Anaphylaxis Program, The University of Toronto, Toronto, ON, Canada

¹¹Department of Allergy and Immunology, Hospital Quironsalud Bizkaia, Erandio, Spain

¹²Centre for Research in Environmental Epidemiology (CREAL), ISGlobAL, Barcelona, Spain

¹³IMIM (Hospital del Mar Research Institute), Barcelona, Spain

¹⁴Universitat Pompeu Fabra (UPF), Barcelona, Spain

¹⁵CIBER Epidemiología y Salud Pública (CIBERESP), Barcelona, Spain

¹⁶Upper Airways Research Laboratory, ENT Department, Ghent University Hospital, Ghent, Belgium

¹⁷International Airway Research Center, First Affiliated Hospital Guangzhou, Sun Yat-sen University, Guangzhou, China

¹⁸Division of ENT Diseases, CLINTEC, Karolinska Institutet, Stockholm, Sweden

¹⁹Department of ENT Diseases, Karolinska University Hospital, Stockholm, Sweden

²⁰Department of Medicine, University of Cape Town, Cape Town, South Africa

²¹Department of Respiratory Medicine, National Institute of Diseases of the Chest and Hospital, Dhaka, Bangladesh

²²Allergology and Clinical Immunology, Carol Davila University of Medicine and Pharmacy, Bucharest, Romania

²³Clinical Emergency Hospital for Children MS Curie, Bucharest, Romania

²⁴Department of Geriatrics, Montpellier University Hospital, Montpellier, France

²⁵EA:2991, Euromov, University Montpellier, Montpellier, France

²⁶Department of Cardiovascular and Thoracic Sciences, Fondazione Policlinico Universitario A Gemelli IRCCS, Università Cattolica del Sacro Cuore, Rome, Italy

²⁷National Heart and Lung Institute, Royal Brompton Hospital and Imperial College London, London, UK

²⁸Woolcock Institute of Medical Research, University of Sydney, Sydney, NSW, Australia

²⁹Woolcock Emphysema Centre and Sydney Local Health District, Glebe, NSW, Australia

³⁰Quebec Heart and Lung Institute, Laval University, Québec City, QC, Canada

³¹Allergy and Clinical Immunology Unit, Department of Medical Sciences, University of Torino and Mauriziano Hospital, Torino, Italy

³²Department of Pulmonary Medicine, Mainz University Hospital, Mainz, Germany

³³Department of Pediatrics, Medical School, Federal University of Minas Gerais, Belo Horizonte, Brazil

³⁴Personalized Medicine Asthma and Allergy Clinic-Humanitas University and Research Hospital, IRCCS-Milano, Milano, Italy

³⁵Allergy Section, Department of Internal Medicine, Hospital Vall d'Hebron and ARADyAL research network, Barcelona, Spain

³⁶Division of Allergy/immunology, University of South Florida, Tampa, FL, USA

³⁷School of Medicine, University CEU San Pablo, Madrid, Spain

³⁸Faculty of Public Health, Medical University - Sofia, Sofia, Bulgaria

³⁹Fundação ProAR, Federal University of Bahia and GARD/WHO Planning Group, Salvador, Brazil

⁴⁰Medical Consulting Czarlewski, Levallois, France

⁴¹Department of Medical Sciences and Public Health and Unit of Allergy and Clinical Immunology, University Hospital "Dulio Casula", University of Cagliari, Cagliari, Italy

⁴²Department of Respiratory Medicine, Wuhan Children's Hospital, Tongji Medical College, Huazhong, University of Science and Technology, Wuhan, Hubei, China

⁴³Pediatric Allergy and Immunology Unit, Children's Hospital, Ain Shams University, Cairo, Egypt

- ⁴⁴Department of Otorhinolaryngology, Academic Medical Centers, AMC, Amsterdam, The Netherlands
- ⁴⁵EUFORÉA, Brussels, Belgium
- ⁴⁶Center for Research in Health Technologies and Information Systems, CINTESIS, Universidade do Porto, Porto, Portugal
- ⁴⁷Allergy Unit, Instituto CUF Porto e Hospital CUF Porto, Porto, Portugal
- ⁴⁸Health Information and Decision Sciences Department - CIDES, Faculdade de Medicina, Universidade do Porto, Porto, Portugal
- ⁴⁹Faculdade de Medicina da Universidade do Porto, Porto, Portugal
- ⁵⁰Department of Allergology, Zhongnan Hospital of Wuhan University, Wuhan, China
- ⁵¹7th Respiratory Medicine Department and Asthma Center, Athens Chest Hospital, Athens, Greece
- ⁵²Department of Pulmonary Diseases, Cerrahpasa Faculty of Medicine, Istanbul University-Cerrahpasa, Istanbul, Turkey
- ⁵³Center of Allergy and Immunology, Georgian Association of Allergology and Clinical Immunology, Tbilisi, Georgia
- ⁵⁴Skin and Allergy Hospital, Helsinki University Hospital, Helsinki, Finland
- ⁵⁵College of Medicine and Health, University of Exeter Medical School, University of Exeter, Exeter, UK
- ⁵⁶Klinik für Kinder- und Jugendmedizin, Kinderzentrum Bethel, Evangelisches Klinikum Bethel EvKB, University Bielefeld, Bielefeld, Germany
- ⁵⁷Department of Pathophysiology and Allergy Research, Medical University of Vienna, Vienna, Austria
- ⁵⁸Service de Pneumologie, Hôpital Bicêtre, Inserm UMR_S999, Université Paris-Sud, Le Kremlin Bicêtre, France
- ⁵⁹National Research Center - Institute of Immunology Federal Medical-Biological Agency of Russia, Moscow, Russia
- ⁶⁰Servicio de Alergia e Immunologia, Clínica Santa Isabel, Buenos Aires, Argentina
- ⁶¹Department of Respiratory Medicine, Ghent University Hospital, Ghent, Belgium
- ⁶²Makerere University Lung Institute, Kampala, Uganda
- ⁶³Departments of Immunology and Dermatology/Allergology, University Medical Center Utrecht, Utrecht, The Netherlands
- ⁶⁴Department of Medicine and Therapeutics, The Chinese University of Hong Kong, Hong Kong, Hong Kong
- ⁶⁵Finnish Institute for Health and Welfare, Helsinki, Finland
- ⁶⁶Department of Immunology and Allergy, Healthy Ageing Research Center, Medical University of Lodz, Lodz, Poland
- ⁶⁷Department of Otorhinolaryngology, Head and Neck Surgery, Semmelweis University, Budapest, Hungary
- ⁶⁸Division of Internal Medicine, Asthma and Allergy, Barlicki University Hospital, Medical University of Lodz, Lodz, Poland
- ⁶⁹Department of Pathology, Faculty of Medicine, Institute of Biomedical Sciences, Vilnius University, Vilnius, Lithuania
- ⁷⁰Clinic of Chest diseases and Allergology, Faculty of Medicine, Institute of Clinical medicine, Vilnius University, Vilnius, Lithuania
- ⁷¹Allergy and Immunology Centre, Pantai Hospital, Kuala Lumpur, Malaysia
- ⁷²University of Medicine and Pharmacy, Hochiminh City, Vietnam
- ⁷³Division Paediatric Allergology, University of Cape Town, Cape Town, South Africa
- ⁷⁴Center of Excellence in Asthma and Allergy, Médica Sur Clinical Foundation and Hospital, México City, Mexico
- ⁷⁵Department of Pulmonary Medicine, CHU Sart-Tilman, and GIGA I3 Research Group, Liege, Belgium
- ⁷⁶Tobacco Control Research Centre, Iranian Anti Tobacco Association, Tehran, Iran
- ⁷⁷Sachs' Children and Youth Hospital, Södersjukhuset, Stockholm, Sweden
- ⁷⁸Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden
- ⁷⁹National Institute of Pneumology M Nasta, Bucharest, Romania
- ⁸⁰Clinic for Pulmonary Diseases, Clinical Center of Serbia, Faculty of Medicine, Serbian Association for Asthma and COPD, University of Belgrade, Belgrade, Serbia
- ⁸¹National Center for Research in Chronic Respiratory Diseases, Tishreen University School of Medicine, Latakia, Syria
- ⁸²Syrian Private University-Damascus, Damas, Syria
- ⁸³Allergy Center, CUF Descobertas Hospital, Lisbon, Portugal
- ⁸⁴Rhinology Unit and Smell Clinic, ENT Department, Hospital Clínic, Barcelona, Spain
- ⁸⁵Clinical and Experimental Respiratory Immunoallergy, IDIBAPS, CIBERES, University of Barcelona, Barcelona, Spain
- ⁸⁶Scientific Centre of Children's Health Under the MoH, Moscow, Russia
- ⁸⁷Russian National Research Medical University Named Pirogov, Moscow, Russia
- ⁸⁸Director of Center of Allergy, Immunology and Respiratory Diseases, Santa Fe, Argentina
- ⁸⁹Argentina Center for Allergy and Immunology, Santa Fe, Argentina
- ⁹⁰Serviço de Pneumologia Hospital Central and Faculdade de Medicina Dr Eduardo Mondelane, Maputo, Mozambique
- ⁹¹Division of Respirology, Department of Medicine, McMaster University, Hamilton, ON, Canada
- ⁹²Firestone Institute for Respiratory Health, St Joseph's Healthcare, Hamilton, ON, Canada
- ⁹³Department of Allergy, Immunology and Respiratory Medicine, Central Clinical School, Monash University, and Alfred Health, Melbourne, Vic., Australia
- ⁹⁴Departments of Medicine and Microbiology, APC Microbiome Ireland, University College Cork, Cork, Ireland
- ⁹⁵National Hospital Organization, Tokyo National Hospital, Tokyo, Japan
- ⁹⁶Department of Otorhinolaryngology, Chiba University Hospital, Chiba, Japan

- ⁹⁷Department of Immunology and Allergology, Faculty of Medicine and Faculty Hospital in Pilsen, Charles University in Prague, Pilsen, Czech Republic
- ⁹⁸Division of Infection, Immunity and Respiratory Medicine, Royal Manchester Children's Hospital, University of Manchester, Manchester, UK
- ⁹⁹Allergy and Respiratory Diseases, Ospedale Policlinico San Martino -University of Genoa, Genoa, Italy
- ¹⁰⁰Division of Allergy and Clinical Immunology, Department of Medicine, Agency of Health ASL Salerno, "Santa Maria della Speranza" Hospital, Salerno, Italy
- ¹⁰¹Department of Pediatrics, Nippon Medical School, Tokyo, Japan
- ¹⁰²Ecole Polytechnique Palaiseau, IRBA (Institut de Recherche bio-Médicale des Armées), Bretigny, France
- ¹⁰³Société de Pneumologie de Langue Française, Espace Francophone de Pneumologie, Paris, France
- ¹⁰⁴University Hospital "Sv Ivan Rilski", Sofia, Bulgaria
- ¹⁰⁵Allergy and Clinical Immunology Unit, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal
- ¹⁰⁶Faculty of Medicine, Institute of Immunology, University of Coimbra, Coimbra, Portugal
- ¹⁰⁷Faculty of Medicine, ICBR - Coimbra Institute for Clinical and Biomedical Research, CIBB, University of Coimbra, Coimbra, Portugal
- ¹⁰⁸Division of Allergy Asthma and Clinical Immunology, Emek Medical Center, Afula, Israel
- ¹⁰⁹Rappaport Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel
- ¹¹⁰Department of Prevention of Environmental Hazards and Allergology, Medical University of Warsaw, Warsaw, Poland
- ¹¹¹Faculty of Medicine, Fundacion Jimenez Diaz, CIBERES, Autonoma University of Madrid, Madrid, Spain
- ¹¹²Centre for Inflammation Research, Child Life and Health, The University of Edinburgh, Edinburgh, UK
- ¹¹³The Usher Institute of Population Health Sciences and Informatics, The University of Edinburgh, Edinburgh, UK
- ¹¹⁴PROMISE Department, University of Palermo, Palermo, Italy
- ¹¹⁵Department of Pediatrics, Hospital Nacional de Niños, San José, Costa Rica
- ¹¹⁶Department of Respiratory Medicine, University Hospital Olomouc, Olomouc, Czech Republic
- ¹¹⁷Allergy and Clinical Immunology Unit, Department of Medical Sciences, University of Torino and Mauriziano Hospital, Torino, Italy
- ¹¹⁸Pulmonary Division, Heart Institute (InCor), Hospital da Clinicas da Faculdade de Medicina da Universidade de Sao Paulo, Sao Paulo, Brazil
- ¹¹⁹Department of Respiratory Medicine, Hvidovre Hospital and University of Copenhagen, Copenhagen, Denmark
- ¹²⁰Faculty of Health Sciences, University of Beira Interior, Covilhã, Portugal
- ¹²¹Department of Immunoallergology, Cova da Beira University Hospital Centre, Covilhã, Portugal
- ¹²²The Hospital for Sick Children, Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada
- ¹²³Department of General ORL, H&NS, Medical University of Graz, ENT-University Hospital Graz, Graz, Austria
- ¹²⁴Health Planning Unit, Department of Social Medicine, Faculty of Medicine, University of Crete, Crete, Greece
- ¹²⁵International Primary Care Respiratory Group International Primary Care Respiratory Group, (IPCRG), Aberdeen, Scotland
- ¹²⁶Airways Disease Section, National Heart and Lung Institute (NHLI), Imperial College London and Royal Brompton Hospital, London, UK
- ¹²⁷Faculty of Medicine, Institute of Clinical Medicine and Institute of Health Sciences, Vilnius University, Vilnius, Lithuania
- ¹²⁸European Academy of Paediatrics (EAP/UEMS-SP), Brussels, Belgium
- ¹²⁹Unit of Geriatric Immunoallergology, University of Bari Medical School, Bari, Italy
- ¹³⁰Pulmonary Environmental Epidemiology Unit, CNR Institute of Clinical Physiology, Pisa, Italy
- ¹³¹CNR Institute for Biomedical Research and Innovation, Palermo, Italy
- ¹³²Sotiria Hospital, Athens, Greece
- ¹³³Department of Otolaryngology, Yong Loo Lin School of Medicine, National University of Singapore, Singapore, Singapore
- ¹³⁴International Primary Care Respiratory Group IPCRG, Aberdeen, Scotland
- ¹³⁵Department of Paediatrics, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, Hong Kong
- ¹³⁶Department of Pulmonology, Celal Bayar University, Manisa, Turkey
- ¹³⁷Universidad Nacional de Villa Maria, Universidad Católica de Córdoba, Córdoba, Argentina
- ¹³⁸University Clinic of Respiratory and Allergic Diseases, Golnik, Slovenia
- ¹³⁹Transylvania University Brasov, Brasov, Romania

Correspondence: Jean Bousquet, CHU Arnaud de Villeneuve, 371 Avenue du Doyen Gaston Giraud, 34295 Montpellier Cedex 5, France.
Email: jean.bousquet@orange.fr

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To the Editor,

A novel strain of human coronaviruses, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), named by the International Committee on Taxonomy of Viruses (ICTV),¹ has recently emerged and caused an infectious disease. This disease is

referred to as the "coronavirus disease 2019" (COVID-19) by the World Health Organization (WHO).²

The US Centers for Disease Control and Prevention (CDC) have proposed that "People with moderate to severe asthma may be at higher risk of getting very sick from COVID-19. COVID-19 can affect

your respiratory tract (nose, throat, lungs), cause an asthma attack and possibly lead to pneumonia and acute respiratory disease.” (May 24, 2020). (<https://www.cdc.gov/coronavirus/2019-ncov/need-extra-precautions/asthma.html>) On the other hand, in the UK, NICE proposes rapid guidelines for severe asthma (<https://www.guidelines.co.uk/covid-19-rapid-guideline-severe-asthma/455275>. article).

An ARIA-EAACI statement has been devised to make recommendations on asthma, and not necessarily on severe asthma, based on a consensus from its members.

It is difficult in many studies to clearly assess the prevalence of asthma on COVID-19 since most patients are older adults and probably have multimorbidities. Most studies do not clarify whether asthmatic patients with COVID-19 have isolated asthma or asthma as a multimorbidity, particularly in the context of hypertension, obesity and diabetes. In particular, obesity is a significant risk factor for COVID-19 and its severity,³ and may be intertwined with asthma.

In some studies, showing data mostly on critically ill patients, there does not appear to be an increased prevalence of asthma.⁴⁻⁷ In Wuhan, the prevalence of asthma in COVID-19 patients was 0.9%, markedly lower than that of the general adult population of this city.⁸ Differently, in New York, among 5,700 hospitalized patients with COVID-19, asthma prevalence was 9% and COPD 4.5%.⁹ In California, 7.4% of the 377 hospitalized patients had asthma or COPD.¹⁰ The US CDC reported that between March 1st and 30th 2020, among COVID-NET hospitals from 99 counties and 14 states (an open source neural network for COVID-19 infection), chronic lung disease (primarily asthma) was the second most prevalent comorbid condition for hospitalized patients aged 18-49 years with laboratory-confirmed COVID-19.¹¹ Among the 17% of COVID-19-positive patients with an underlying history of asthma, the incidence was at its highest in younger adults (27% in the 18- to 49-year-old group). The UK experience on over 20, 133 hospitalized cases shows that 14% of admissions were patients with asthma.¹² In the OpenSAFELY Collaborative Study (UK), an increased risk of severe COVID-19, including death, was found in patients with asthma, particularly related with a recent use of oral corticosteroid.¹³ A review with all identified studies up to 5 May 2020 is available.¹⁴ However, low socioeconomic status, obesity, non-white ethnicity, chronic respiratory disease and diabetes had stronger signals.

Some anti-asthma medications, such as ciclesonide, might have a beneficial effect on COVID-19.¹⁵

Thus, whether patients with asthma are at a higher or lower risk of acquiring COVID-19 may depend on geography, age, other multimorbidities, different air quality,¹⁶ genetic predispositions, ethnicity, social behaviour, access to health care or other factors. Moreover, the current information is obtained mainly from hospitalization or intensive care unit data. Real-life data in a non-selected population of asthmatics are needed to better understand the links between asthma and SARS-Cov-2 in terms of both incidence and severity.

Asthma does not seem to be a risk factor for severe COVID-19 but patients treated with oral corticosteroids may be at a higher risk

of severe COVID-19.¹⁴ However, a large study is needed to fully appreciate the relationship between COVID-19 and severe asthma.

According to the IPCRG (International Primary Care Respiratory Group), patients are still struggling to differentiate their symptoms between asthma flare-ups and COVID-19. They may therefore delay seeking care for asthma or COVID-19. Interestingly, clarity does not appear to have improved as the weeks have passed. People have recurrences or waves of repeated symptoms, and it is difficult to understand whether the symptoms are related to an asthma exacerbation or to COVID-19.

According to the IPCRG, many clinicians tend to prescribe antibiotics to people who they believe are having asthma exacerbations “just to be safe.” They focus on the potential infection element of the trigger more than the asthma management itself. It would seem that COVID-19 might exacerbate this behaviour, not improve it.

In areas where COVID-19 is prevalent, GPs are still very concerned about oral—and, to a certain degree, inhaled—corticosteroids, possibly because they use remote models of care. They are reluctant to prescribe higher doses of ICS or OCS as they fear they cannot tell the difference between a flare-up and COVID-19.

The extent of expression in the upper and lower airways of the SARS-CoV-2 entry receptors, angiotensin-converting enzyme 2 (ACE2) and TMPRSS2, might impact the clinical severity of COVID-19. ACE-2 was found to be decreased in patients with allergic asthma¹⁷ or in those receiving inhaled corticosteroids.¹⁸ These data suggest that this expression may be a potential contributor, among several other factors, to reduced COVID-19 severity in patients with T2 inflammation.^{17,19} However, ACE-2 expression in asthma patients was increased in African Americans, in males and in association with diabetes.

Finally, a recent study which analysed the nasal transcriptome of 695 children suggested that the strongest determinants of airway ACE2 and TMPRSS2 expression are T2 inflammation and viral-induced interferon inflammation. However, this study specifically showed that T2 inflammation (via IL-13) impacted differentially on ACE2 and TMPRSS2, with a T2-high phenotype being associated with a highly significant decrease in the former and a significant decrease in the latter receptor. Thus, although SARS-CoV-2-specific analyses and experiments are lacking, the differential effects of T2-inflammation on ACE2 and TMPRSS2 reported in this study warrant further research on whether T2-high and T2-low asthma phenotypes may be associated with differential susceptibility to severe COVID-19.

The first author developed seven recommendations that were sent for comment to 105 experts around the world. 69 answers were received within 48 hours, and the comments were considered. Where experts suggested modification of the recommendations, a discussion was initiated and recommendations modified until consensus was reached. After these modifications, a total of 9 recommendations were proposed for a second round. In the second round, 145 experts were invited to comment on and approve or reject the recommendations. 78 answers were received within 48 hours and, when an agreement of over 80/100 was reached, the question was included in the statement.

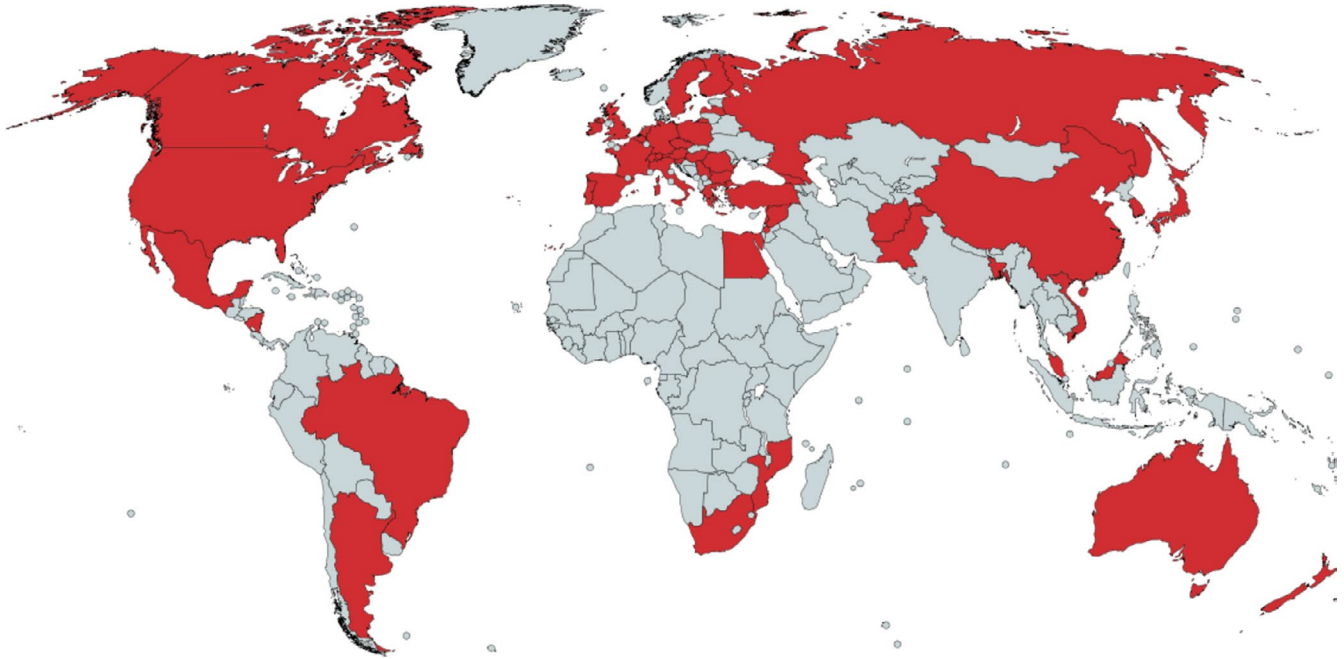


FIGURE 1 Geographic representation of the experts

The same approach was used for the research questions. Two research needs were dropped.

The geographic distribution of the experts is given in Figure 1. They were from 43 countries.

ARIA-EAACI statement (Table 1).

ARIA-EAACI research questions (Table 2).

This view is pragmatic, cautious and based upon expert opinion. However, it is likely to require modifications as further evidence is

- 1 In areas where COVID-19 is prevalent, screening protocols for COVID-19 should be applied to anyone having worsening respiratory symptoms, and personal protective equipment should be used.
- 2 In areas where COVID-19 is prevalent, lung function testing procedures should be postponed if not deemed absolutely necessary; portable personal devices measuring PEF and FEV1 can be used in the meantime to monitor asthma control using the telemedicine approach.
- 3 In accordance with the Global Initiative for Asthma (GINA) (<https://ginasthma.org/recommendations-for-inhaled-asthma-controller-medications/>), patients with asthma should not stop their prescribed inhaled corticosteroid controller medication (or prescribed oral corticosteroids). Stopping inhaled corticosteroids may have serious consequences.
- 4 Long-term oral corticosteroids may sometimes be required to treat severe asthma, and it may be dangerous to stop them suddenly (GINA).
- 5 Oral steroids should continue to be used to treat severe asthma exacerbations.
- 6 In patients infected by SARS-CoV-2 (symptomatic or asymptomatic), nebulization (which increases the risk of deposition of the virus into the lower airways) should be replaced by spacers of large capacity.
- 7 In accordance with the NICE, in non-SARS-CoV-2 infected patients, we propose(<https://www.nice.org.uk/guidance/ng166/chapter/3-Treatment#patients-having-biological-treatment>):
 - To continue biologics because there is no evidence that biological therapies for asthma suppress immunity
 - If the patient usually attends a hospital for biological treatments, to think about if he/she can be trained to self-administer or could be treated at a community clinic or at home
 - To carry out routine monitoring of biological treatment remotely if possible
- 8 In SARS-CoV-2-infected patients, in accordance with the EAACI, we propose to cease the treatment until resolution of the disease is established. Thereafter, the administration of the biological should be re-initiated.

TABLE 1 ARIA-EAACI statement

TABLE 2 ARIA-EAACI research questions

Real-world studies need to be carried out on a large number of unselected patients to assess

- 1 Impact of COVID-19 on asthma control
- 2 Impact of COVID-19 respiratory symptoms on severe asthma
- 3 Impact of severe asthma on COVID-19 occurrence and/or severity of pneumonia
- 4 Impact of multimorbidities on asthmatic patients for the control of asthma during COVID-19
- 5 Serologic studies should be performed to assess whether seroconversion and its duration differ in asthmatic and non-asthmatic subjects
- 6 The phenotype of asthma (allergic, neutrophilic, age...) should be studied
- 7 In adult patients, studies should clarify whether asthmatic patients with COVID-19 have isolated asthma or asthma in the context of multimorbidity, particularly in the context of high blood pressure, obesity and diabetes mellitus
- 8 Role of pollen season on COVID-19 severity

gathered. These recommendations are conditional and should be adapted regularly on the basis of evolving clinical evidence.

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CONFLICTS OF INTEREST

IA reports and Associate Editor of Allergy. CA reports grants from Allergopharma, Idorsia, Swiss National Science Foundation, Christine Kühne-Center for Allergy Research and Education, European Commission's Horizon's 2020 Framework Programme, Cure, Novartis Research Institutes, Astra Zeneca, Scibase, advisory role in Sanofi/Regeneron. IA reports personal fees from Mundipharma, Roxall, Sanofi, MSD, Faes Farma, Hikma, UCB, Astra Zeneca, Stallergenes, Abbott, Bial. EB is a member of the Science Committee and Board of the Global Initiative for Asthma (GINA). SBA reports grants from TEVA, personal fees from TEVA, AstraZeneca, Boehringer Ingelheim, GSK, Sanofi, Mylan. JPB reports grants from AstraZeneca, Boston Scientific, GSK, Hoffman La Roche, Ono Pharma, Novartis, Sanofi, Takeda, Boehringer-Ingelheim, Merck, personal fees from AstraZeneca, GSK, Merck, Metapharm, Novartis, Takeda, other from AstraZeneca, Boehringer-Ingelheim, GSK, Merck, Novartis. JB reports personal fees from Chiesi, Cipla, Hikma, Menarini, Mundipharma, Mylan, Novartis, Purina, Sanofi-Aventis, Takeda, Teva, Uriach, other from KYomed-Innov. RB reports grants to Mainz University and personal fees from Boehringer Ingelheim, GlaxoSmithKline, Novartis, and Roche, as well as personal fees from AstraZeneca, Chiesi, Cipla, Sanofi, and Teva. VC reports personal fees from ALK, Allergopharma, Allergy Therapeutics, Diater, LETI, Thermo Fisher, Stallergenes. RSC reports grants from NIAID, CoFAR, Aimmune, DBV Technologies, Astellas, Regeneron, an Advisory member for Alladapt, Genentech, Novartis, and receives personal fees from Before Brands. AC reports grants and personal fees from GSK, SANOFI, Boehringer-Ingelheim, Astrazeneca, Mantecorp, MYLAN, Novartis, personal fees and non-financial support from CHIESI. SdG reports personal fees from AstraZeneca, Chiesi, Menarini, grants and personal fees from GSK, Novartis. DH reports personal fees from AstraZeneca, Chiesi, GSK, Pfizer, personal fees

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AstraZeneca, Chiesi, Boehringer Ingelheim. IT reports grants from GSK Hellas, ELPEN, personal fees from Boehringer Ingelheim, Novartis, Astra Zeneca, GSK. TZ reports Organizational affiliations: Committee member: WHO-Initiative "Allergic Rhinitis and Its Impact on Asthma" (ARIA); Member of the Board: German Society for Allergy and Clinical Immunology (DGAKI); Head: European Centre for Allergy Research Foundation (ECARF); President: Global Allergy and Asthma European Network (GA²LEN); Member: Committee on Allergy Diagnosis and Molecular Allergology, World Allergy Organization (WAO). The other authors have no COI to declare.

ORCID

Cezmi A. Akdis  <https://orcid.org/0000-0001-8020-019X>
 Oliver Pfaar  <https://orcid.org/0000-0003-4374-9639>
 Kari C. Nadeau  <https://orcid.org/0000-0002-2146-2955>
 Thomas Eiwegger  <https://orcid.org/0000-0002-2914-7829>
 Claus Bachert  <https://orcid.org/0000-0003-4742-1665>
 Karl-Christian Bergmann  <https://orcid.org/0000-0002-0306-9922>
 Mateo Bonini  <https://orcid.org/0000-0002-3042-0765>
 Louis-Philippe Boulet  <https://orcid.org/0000-0003-3485-9393>
 Victoria Cardona  <https://orcid.org/0000-0003-2197-9767>
 Thomas Casale  <https://orcid.org/0000-0002-3149-7377>
 Mübeccel Akdis  <https://orcid.org/0000-0003-0554-9943>
 Alvaro A. Cruz  <https://orcid.org/0000-0002-7403-3871>
 Wytske J. Fokkens  <https://orcid.org/0000-0003-4852-229X>
 Maia Gotua  <https://orcid.org/0000-0003-2497-4128>
 Tari Haahtela  <https://orcid.org/0000-0003-4757-2156>
 Eckard Hamelmann  <https://orcid.org/0000-0002-2996-8248>
 Dmitry Kudlay  <https://orcid.org/0000-0003-1878-4467>
 Michael Levin  <https://orcid.org/0000-0003-2439-7981>
 Desiree Larenas-Linnemann  <https://orcid.org/0000-0002-5713-5331>
 Mario Morais-Almeida  <https://orcid.org/0000-0003-1837-2980>
 Ken Ohta  <https://orcid.org/0000-0001-9734-4579>
 Nikos G. Papadopoulos  <https://orcid.org/0000-0002-4448-3468>
 Gianni Passalacqua  <https://orcid.org/0000-0002-5139-3604>
 Vincenzo Patella  <https://orcid.org/0000-0001-5640-6446>
 Joaquin Sastre  <https://orcid.org/0000-0003-4689-6837>
 Peter-Valentin Tomazic  <https://orcid.org/0000-0001-6445-4800>
 Sanna Toppila-Salmi  <https://orcid.org/0000-0003-0890-6686>
 Gary W. K. Wong  <https://orcid.org/0000-0001-5939-812X>
 Torsten Zuberbier  <https://orcid.org/0000-0002-1466-8875>
 Ioana Agache  <https://orcid.org/0000-0001-7994-364X>

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