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

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), 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). 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, 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. 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. Differently, in New York, among 5,700 hospitalized patients with COVID-19, asthma prevalence was 9% and COPD 4.5%. In California, 7.4% of the 377 hospitalized patients had asthma or COPD. 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. 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. 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. A review with all identified studies up to 5 May 2020 is available. 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.

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, 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. 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 asthma\textsuperscript{17} or in those receiving inhaled corticosteroids.\textsuperscript{18} These data suggest that this expression may be a potential contributor, among

| FIGURE 1 | Geographic representation of the experts |

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 |

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\textsuperscript{17} Zmora N, et al. (2020) [2020].

\textsuperscript{18} De Santis V, et al. (2020) [2020].
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.

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

**ACKNOWLEDGMENT**
Open access funding enabled and organized by Projekt DEAL.

**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. JB 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, Manteorp, 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 and non-financial support from Boehringer Ingelheim, Novartis. TE reports other from DBV, Regeneron, grants from Innovation Fund Denmark and Co-I or scientific lead in three investigator initiated oral immunotherapy trials supported by the Allergy and Anaphylaxis Program Sickkids and serve as associate editor for Allergy, Advisory board ALK. JF reports personal fees from AstraZeneca, GSK, undipharma, grants and personal fees from Novartis. MG reports grants and personal fees from Elpen, Novartis, Menarini, grants from Galapagos, personal fees from BMS, MSD. TH reports personal fees from GSK, Mundipharma, OrionPharma. MH reports personal fees and non-financial support from GlaxoSmithKline, personal fees from AstraZeneca, Novartis, Roche, Sanofi, Teva. JCI reports personal fees from Faes Farma, Eurofarma Argentina, other from Laboratorios Casasco, Sanofi. GJ reports grants from AstraZeneca, Chiesi, personal fees from Bayer, Eureka vzw, Teva, grants and personal fees from GlaxoSmithKline. MJ reports personal fees from ALK-Abello, Allergopharma, Stallergenes, Anergis, Allergy Therapeutics, Circassia, Leti, Biomay, from HAL, Astra-Zeneka, GSK, Novartis, Teva, Vectura, UCB, Takeda, Roche, Janssen, Medimmune, Chiesi. LK reports grants and personal fees from Allergopharma, LETI Pharma, MEDA/Mylan, Sanofi, personal fees from HAL Allergy, Allergy Therapeutics, grants from ALK Abelló, Stallergenes, Quintiles, ASIT biotech, grants from Lofarma, AstraZeneca, GSK, Immunotek and Membership: AeDA, DGHNO, Deutsche Akademie für Allergologie und klinische Immunologie, HNO-BV GPA, EAACI. PK reports personal fees from Astra, Boehringer Ingelheim, Berlin Chemie Menarini, GSK, Lekam, Novartis, Polpharma, Mylan, Orion, Teva, Adamed. VK reports personal fees from GSK, non-financial support from StallergenGreer, AstraZeneca, Norameda, DIMUNA. 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KN reports grants and other from NIAID, FARE, personal fees and other from Regeneron, grants from EAT, other from Sanofi, Astellas, Nestle, BeforeBrands, Alladapt, ForTra, Genentech, Alimune Therapeutics, DBV Technologies, personal fees from AstraZeneca, ImmuneWorks, Cour Pharmaceuticals, grants from Allergeni, Ukko Pharma, Novartis, AnaptyxBio, Adare Pharmaceuticals, Stallergenes-Greer, NHLBI, NIEHS, EPA, WAO Center of Excellence, Iggenix, Probio, Vedanta, Centecor, Seed, Immune Tolerance Network, NIH.; In addition, Dr Nadeau has a patent Inhibition of Allergic Reaction to Peanut Allergen using an IL-33 Inhibitor pending, a patent Special Oral Formula for Decreasing Food Allergy Risk and Treatment for Food Allergy pending, a patent Basophil Activation Based Diagnostic Allergy Test pending, a patent Granulocyte-based methods for detecting and monitoring immune system disorders pending, a patent Methods and Assays for Detecting and Quantifying Pure Subpopulations of White Blood Cells in Immune System Disorders pending, a patent Mixed Allergen Compositions and Methods for Using the Same pending, and a patent Microfluidic Device and Diagnostic Methods for Allergy Testing Based on Detection of Basophil Activation pending. YO reports personal fees from Shionogi Co., Ltd., Torii Co., Ltd., GSK, MSD, Eizai Co., Ltd., grants and personal fees from Kyorin Co., Ltd., Tiho Co., Ltd., grants from Yakuruto Co., Ltd., Yamada Bee Farm. ROB reports grants and personal fees from AstraZeneca, GSK, grants from Novartis, Medimmune, Bayer. YO reports personal fees from Shionogi Co., Ltd., Torii Co., Ltd., GSK, MSD, Eizai Co., Ltd., grants and personal fees from Kyorin Co., Ltd., Tiho Co., Ltd., grants from Yakuruto Co., Ltd., Yamada Bee Farm, outside the submitted work. NP reports personal fees from Novartis, Nutricia, HAL, MENARINI/FAES FARMA, SANOFI, MYLAN/MEDA, BIOMAY, AstraZeneca, GSK, MSD, ASIT BIOTECH, Boehringer Ingelheim, grants from Gerolymatos International SA, Capricare. OP reports grants and personal fees from Anergis SA, ALK-Abelló, Allergopharma, Stallergenes Greer, HAL Allergy Holding BV/HAL Allergie GmbH, Bencard Allergie GmbH/Allergy Therapeutics, Lofarma, ASIT Biotech Tools SA, Laboratorios LETI/LETI Pharma, grants from Biomay, Glaxo Smith Kline Circassia, personal fees from MEDA Pharma/MYLAN, Mobile Chamber Experts (a GA2LEN Partner), Indoor Biotechnologies, Astellas Pharma Global, EUFOREA, ROXALL, NOVARTIS, SANOFI AVENTIS, Med Update Europe GmbH, streamdup! GmbH. FP reports sanofi, novartis, teva, astrazeneca, glaxosmithkline, menarini, mundipharma, guidotti, malesci, chiesi, valeas, allergy therapeutics, almirall, personal fees from boehringer ingelheim. FR reports personal fees from AstraZeneca, Novartis, Lusomedicamenta, Sanofi, GSK. JS reports other from MEDA, grants and personal fees from SANOFI, personal fees from GSK, NOVARTIS, ASTRA ZENEA, MUNDIPHARMA, FAES FARMA. JSchwarze reports personal fees from MYLAN, outside the submitted work. ASheikh reports support of the Asthma UK Centre for Applied Research. RS reports grants from São Paulo Research Foundation, MSD, grants and personal fees from Novartis, grants, personal fees and non-financial support from 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.

Jean Bousquet1,2,3,4
Marek Juteł5
Cezmi A. Akdis6

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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, Wroclaw Medical University and ALL-MED Medical Research Institute, Wroclaw, 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 Bizaia, 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 Epidemiologia 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, Giebe, 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 “Duilio Casula”, University of Cagliari, Cagliari, Italy
42Department of Allergology, Zhongnan Hospital of Wuhan University, Wuhan, China
43Pediatric Allergy and Immunology Unit, Children’s Hospital, Ain Shams University, Cairo, Egypt
44Department of Otorhinolaryngology, Academic Medical Centers, AMC, Amsterdam, The Netherlands
45EUFOREA, Brussels, Belgium
46Center for Research in Health Technologies and Information Systems, CINTESIS, Universidade do Porto, Porto, Portugal
Division of Allergy and Clinical Immunology, Department of Medicine, Agency of Health ASL Salerno, “Santa Maria della Speranza” Hospital, Salerno, Italy

Department of Pediatrics, Nippon Medical School, Tokyo, Japan

Allergy and Clinical Immunology Unit, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal

Faculty of Medicine, Institute of Immunology, University of Coimbra, Coimbra, Portugal

Division of Allergy Asthma and Clinical Immunology, Emek Medical Center, Afula, Israel

Rappaport Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel

Department of Prevention of Environmental Hazards and Allergology, Medical University of Warsaw, Warsaw, Poland

Faculaty of Medicine, Fundacion Jimenez Diaz, CIBERES, Autonoma University of Madrid, Madrid, Spain

Centre for Inflammation Research, Child Life and Health, The University of Edinburgh, Edinburgh, UK

The Usher Institute of Population Health Sciences and Informatics, The University of Edinburgh, Edinburgh, UK

PROMISE Department, University of Palermo, Palermo, Italy

Department of Pediatrics, Hospital Nacional de Niños, San José, Costa Rica

Department of Respiratory Medicine, University Hospital Olomouc, Olomouc, Czech Republic

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

Pulmonary Division, Heart Institute (InCor), Hospital da Clinicas da Faculdade de Medicina da Universidade de Sao Paulo, Sao Paulo, Brazil

Department of Respiratory Medicine, Hvidovre Hospital and University of Copenhagen, Copenhagen, Denmark

Faculty of Health Sciences, University of Beira Interior, Covilhã, Portugal

Department of Immoalloergology, Cova da Beira University Hospital Centre, Covilhã, Portugal

The Hospital for Sick Children, Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada

Department of General ORL, H&NS, Medical University of Graz, ENT-University Hospital Graz, Graz, Austria

Health Planning Unit, Department of Social Medicine, Faculty of Medicine, University of Crete, Crete, Greece

International Primary Care Respiratory Group International Primary Care Respiratory Group, (IPCRG), Aberdeen, Scotland

Airways Disease Section, National Heart and Lung Institute (NHLI), Imperial College London and Royal Brompton Hospital, London, UK

Faculty of Medicine, Institute of Clinical Medicine and Institute of Health Sciences, Vilnius University, Vilnius, Lithuania

European Academy of Paediatrics (EAP)/UEMS-SP, Brussels, Belgium

Unit of Geriatric Immunology, University of Bari Medical School, Bari, Italy

Pulmonary Environmental Epidemiology Unit, CNR Institute of Clinical Physiology, Pisa, Italy

CNR Institute for Biomedical Research and Innovation, Palermo, Italy

Sotiria Hospital, Athens, Greece

Department of Otolaryngology, Yong Loo Lin School of Medicine, National University of Singapore, Singapore

International Primary Care Respiratory Group IPCRG, Aberdeen, Scotland

Department of Paediatrics, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, Hong Kong

Department of Pulmonology, Celal Bayar University, Manisa, Turkey

Universidad Nacional de Villa Maria, Universidad Católica de Córdoba, Córdoba, Argentina

University Clinic of Respiratory and Allergic Diseases, Gornik, Slovenia

Transylvania 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

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
Kari-Christian Bergmann https://orcid.org/0000-0002-0306-9922
Mateo Bonini https://orcid.org/0000-0002-3042-0765
Louis-Phillippe 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
3. Wadhera RK, Wadhera P, Gaba P, et al. Variation in COVID-19 hospitalizations and deaths across New York City boroughs. JAMA. 2020;323(20):2052-2059.

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