the current acute asthma treatment paradigm (6). The longer half-life of benralizumab and the harms of systemic corticosteroids may tip the cost–benefit assessment in favor of benralizumab.

We agree that more work is needed before benralizumab becomes an option for the management of asthma attacks. Nevertheless, the rapidity of eosinophil depletion certainly makes it an exciting prospect. We look forward to the results of our clinical trial to examine this idea (clinicaltrials.gov ID: NCT04098718). ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

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Erratum: COVID-19–related Genes in Sputum Cells in Asthma: Relationship to Demographic Features and Corticosteroids

Our article, published in the July 1, 2020, issue of the *Journal* (1), contained an error in the number of healthy control subjects. The paper reported on 330 asthma participants in the SARP-3 (NHLBI Severe Asthma Research Program-3) cohort and 79 healthy control subjects (57 recruited by the University of California San Francisco [UCSF] Airway Clinical Research Center and 22 recruited by SARP-3). We recently discovered that a coding error resulted in sputum cell RNA from 47 mild asthma patients being included in the UCSF healthy subject group. To correct the error, we removed the 47 mild asthma patients and reanalyzed the data. After performing the reanalysis including the 22 healthy subjects from SARP and 10 healthy subjects from UCSF (total of 32 healthy controls) (revised Table 1), we found that our study conclusions remain the same. As illustrated in revised Figures 1 and 2, sputum cell gene expression for COVID-19–related genes (ACE2 [angiotensin-converting enzyme 2] and TMPRSS2 [transmembrane protease serine 2]) are not significantly different in asthma and health (revised Figure 1A and 1B), and sputum cell gene expression for ACE2 and TMPRSS2 are significantly correlated with one another (revised Figure 2A). The reanalysis shows that the *P* value for the increase in asthma for sputum cell ICAM1 expression (a comparator/control gene) compared with health increased from 0.005 to 0.09 (revised Figure 1C). The main data for the paper, as originally presented in Figures 3 and 4 and which relied on data analyses that were restricted to the asthma cohort, do not need correction.

Table 1. Demographic Features of Subjects with Asthma and Healthy Control Subjects

Characteristic	Healthy (*n* = 32)	Asthma (*n* = 330)	*P* Value
Age, yr	38.8 (13.3)	48.5 (13.8)	<0.001
Sex, F, *n* (%)	24 (75)	230 (69)	0.53
Race, *n* (%)*			0.002
AIAN	0 (0)	2 (1)	
Asian	6 (19)	13 (4)	
African American	4 (13)	77 (23)	
White	18 (58)	217 (66)	
Native Hawaiian or other Pacific Islander	0 (0)	0 (0)	
Mixed race	3 (10)	23 (7)	
BMI, kg/m^2	25.9 (5.6)	32.4 (8.7)	<0.001
Spirometry†			
FEV_1, % predicted	96.2 (10.1)	72.8 (19.3)	<0.001
FVC, % predicted	97.2 (12.8)	85.2 (17.0)	<0.001
FEV_1%/FVC%	99.0 (5.4)	84.5 (12.1)	<0.001

Definition of abbreviations: AIAN = American Indian and Alaska Native; BMI = body mass index.
Data are shown as mean (SD) unless otherwise noted.
*AIAN patients are not included in the mixed effects models because only 2 patients identified as AIAN. One healthy control subject did not answer the race questionnaire.
†Spirometry values are before administration of bronchodilator medications.

We have made several additional changes throughout the article to reflect the corrected data. For the convenience of the readers, the *Journal* has replaced the online version of the article with a corrected version (a redlined version showing the changes may be accessed from the Supplements tag at the top of the HTML view of the article).

We apologize to the readership for these errors. ■

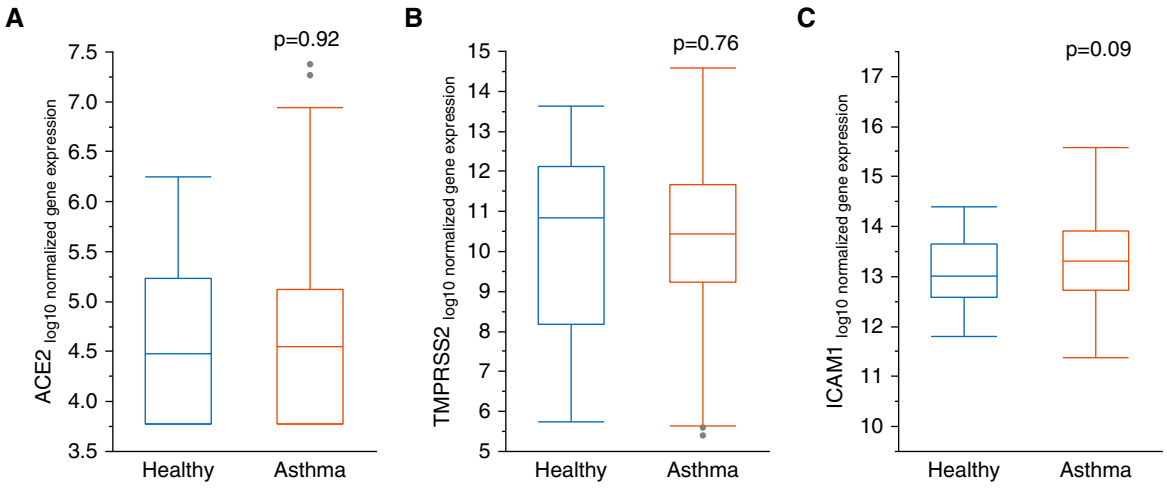

Figure 1. Sputum gene expression at the initial study visit in participants with asthma (*n* = 330) and healthy participants (*n* = 32). (*A* and *B*) No difference in severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)–related genes, ACE2 (angiotensin-converting enzyme 2) and TMPRSS2 (transmembrane protease serine 2), between participants with asthma and healthy participants. (*C*) Gene expression for the rhinovirus binding protein ICAM1 (intercellular adhesion molecule 1) trended higher in asthma participants compared with healthy participants.

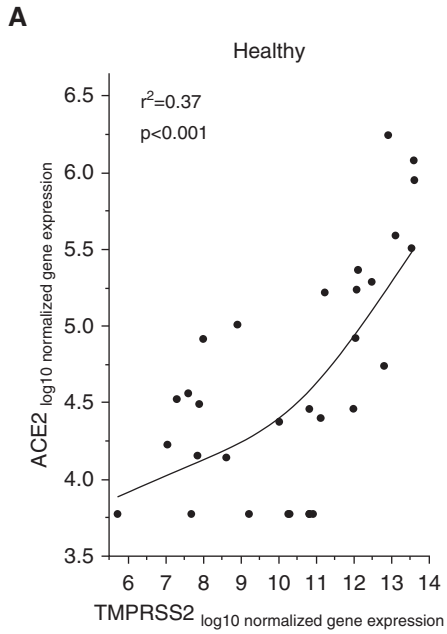

Figure 2A. Sputum gene expression of ACE2 (angiotensin-converting enzyme 2) and TMPRSS2 (transmembrane protease serine 2) is strongly correlated in healthy participants. Best fit line was fitted using a cubic smoothing spline.

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Erratum: A Chronic Obstructive Pulmonary Disease Susceptibility Gene, *FAM13A*, Regulates Protein Stability of β-Catenin

The *Journal* has been alerted to errors in two figures in the article by Jiang and colleagues (1), published in the July 15, 2016, issue.

In Figure 1A, an incorrect image was used for the lower left panel (Rb IgG, Goat IgG). In addition, the panels in the bottom row included scale bars that were incorrectly labeled; these should be 20 μm instead of 40 μm. A corrected version of the figure is included here.

In Figure E9B in the online supplement to the article, a duplicate image of the lower right panel ($Fam13a^{-/-}$, PKF118–310) was inadvertently used for the lower left panel ($Fam13a^{+/+}$, PKF118–310). A revised version of Figure E9B is included here that includes the correct version of the lower left panel.

The authors apologize to the *Journal's* readers for any confusion caused by these errors. ■

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