in the writing of the report; nor in the decision to submit the article for publication.

Brett G. Toelle1,2
Frances L. Garden3,4
Peter B. McIntyre5,6,7
Nicholas Wood5,6,7
Guy B. Marks1,3,4

1Woolcock Institute of Medical Research, The University of Sydney, Sydney, NSW, Australia
2Sydney Local Health District, Sydney, NSW, Australia
3South Western Sydney Clinical School, University of New South Wales, Sydney, NSW, Australia
4Ingham Institute of Applied Medical Research, Sydney, NSW, Australia
5National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, Westmead, NSW, Australia
6The Children’s Hospital at Westmead, Sydney, NSW, Australia
7The University of Sydney, Sydney, NSW, Australia

Correspondence
Brett G. Toelle, Woolcock Institute of Medical Research, Box M77 Missenden Road Post Office, Camperdown, NSW 2050, Australia.
Email: brett.toelle@woolcock.org.au

Editor: Rachel Peters

ORCID
Brett G. Toelle https://orcid.org/0000-0002-7375-0019

REFERENCES
1. Robinson D, Humbert M, Buhl R, et al. Revisiting Type 2-high and Type 2-low airway inflammation in asthma: current knowledge and therapeutic implications. Clin Exp Allergy. 2017;47(2):161-175.
2. Mullins RJ, Dear KB, Tang ML. Time trends in Australian hospital anaphylaxis admissions in 1998–1999 to 2011–2012. J Allergy Clin Immunol. 2015;136(2):367-375.
3. Garden FL, Toelle BG, Mihrshahi S, et al. Cohort profile: the childhood asthma prevention study (CAPS). Int J Epidemiol. 2018;47(6):1736k.
4. Marks GB, Mihrshahi S, Kemp AS, et al. Prevention of asthma during the first 5 years of life: a randomized controlled trial. J Allergy Clin Immunol. 2006;118(1):53-61.
5. Toelle BG, Ng KK, Crisafulli D, et al. Eight-year outcomes of the childhood asthma prevention study. J Allergy Clin Immunol. 2010;126(2):388-389, 389 e381–383.
6. Toelle BG, Garden FL, Ng KK, et al. Outcomes of the Childhood Asthma Prevention Study At 11.5 years. J Allergy Clin Immunol. 2013;132(5):1220-1222.
7. Torvaldsen S, Hull BP, McIntyre PB. Using the Australian childhood immunisation register to track the transition from whole-cell to acellular pertussis vaccines. Commun Dis Intell. 2002;26(4):581-583.
8. Venter C, Stowe J, Andrews NJ, Miller E, Turner PJ. No association between atopic outcomes and type of pertussis vaccine given in children born on the Isle of Wight 2001–2002. J Allergy Clin Immunol Pract. 2016;4(6):1248-1250.
9. Mrozek-Budzyn D, Majewska R, Kieltyka A, Augustyniak M. Whole-cell pertussis vaccine (DTwP) has no influence on allergic diseases and atopic sensitization in children. Postepy Dermatol Alergol. 2018;35(4):381-386.
10. Estcourt MJ, Campbell DE, Gold MS, et al. Whole cell pertussis vaccination and decreased risk of IgE-mediated food allergy: a nested case-control study. J Allergy Clin Immunol Pract. 2020;8(6):2004-2014.
11. Australian New Zealand clinical trial registry. https://anzctr.org.au/Trial/Registration/TrialReview.aspx?ACTRN=12617000065392. Updated 09/03/2019. Accessed 24/01/2020.

SUPPORTING INFORMATION
Additional supporting information may be found online in the Supporting Information section.

African American children are at higher risk of COVID-19 infection

To the Editor,
Infection by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the viral etiology of the novel coronavirus disease 2019 (COVID-19), was first reported in Wuhan, China, in late 2019. Peculiarly, the virus has not caused significant impact on pediatric populations, unlike other coronaviruses.1 Children comprise only 1.7% of COVID-19-positive cases in the United States.2 Furthermore, children are noted to have a milder disease course.3,4
However, much is unknown about the age, gender, and race risk factors for COVID-19 among children. There has been recent evidence suggestive of higher rates of COVID-19 and related fatality rates in African American adult communities around the United States. However, there are limited data, to our knowledge, whether any race or ethnicity group is at higher risk of COVID-19 infection in children.

Despite COVID-19 causing significant respiratory symptoms, asthma as a possible risk factor for COVID-19 has not been studied in pediatric populations. In this study, we aimed to investigate the demographic risk factors for COVID-19 and the association of pre-existing asthma in a pediatric series evaluated at a COVID-19 clinic in the city of Chicago, a hotspot for COVID-19 infection.

The study was approved by the Institutional Review Board of Rush University Medical Center.

All children (<18 years old) who were evaluated by the University COVID-19 Telemedicine Clinic between March 12 and April 20, 2020, and tested for SARS-CoV-2 by real-time polymerase chain reaction were enrolled in the study. As part of this evaluation, children were screened by a healthcare provider for fever, cough, dyspnea, myalgia, sore throat, and anosmia. Children were tested for COVID-19 if their history included any of these symptoms. They were also asked about potential risk factors in the history which included pre-existing asthma. This information was recorded as part of their telemedicine visit. The demographic and asthma status for all these children were recorded. The terminology and categories used for race are based on the National Institutes of Health recommendation of reporting 5 racial categories and 2 ethnic categories (Hispanic and non-Hispanic) (National Institutes of Health Policy Notice No. NOT-OD-01-053).

Initial comparisons between groups were performed applying either the chi-square test or analysis of variance (ANOVA) tests. Logistic regression was used to test whether demographic factors as well as comorbid conditions influenced results. We used SPSS (IBM SPSS Statistics for Windows, Version 22.0) for statistical analysis. Differences were considered statistically significant at $P \leq .05$.

Over a 5-week time period from March to April 2020, 474 children were evaluated and tested for COVID-19 at our facility. 53.8% of the series were male, and the mean (standard deviation [SD]) of age in the series was 5.11 (5.83) years. In terms of race, 25.1% of all tested children were non-Hispanic white, 43.2% were African American (AA), 24.7% were Hispanic, and 1.5% were Asian. The remaining children were identified as other races.

Among the 474 included cases, 5.2% of children were found to be positive for COVID-19. The mean age of COVID-19–positive children was significantly higher than those tested negative. The mean (SD) was 9.72 (7.13) years vs 4.85 (5.66) years in positive- and negative-tested children, $P < .0001$. Gender was not associated with positive test for COVID-19: 60% of positive-tested vs 53.5% of negative-tested children were male, $P = .33$.

Minority racial/ethnic groups were significantly associated with COVID-19–positive test results. Compared with non-Hispanic whites, AA children had a significantly higher rate of positive test: 6.8% of AA children vs 1.7% of white children tested positive for COVID-19, $P = .046$. Furthermore, Hispanic children had a trend toward higher rate of positive COVID-19 test compared with their white non-Hispanic counterparts; 6.6% vs 1.7% positive-tested in Hispanics vs non-Hispanic whites, $P = .067$. We applied a logistic regression model including age, gender, and race to assess the possible intervariable effects. The results showed that AA race and higher age were risk factors for positive COVID-19 test even after adjusting for all demographics (Table 1).

Among the positive COVID-19 cases, 20% were admitted to the hospital and 12% needed to be admitted to pediatric intensive care unit (PICU). Only one child was intubated. 80% of hospitalized and all PICU-admitted children were AA.

In this series, 10.3% of all children who were tested for COVID-19 had pre-existing asthma. There was no difference in the rate of pre-existing asthma between children who tested positive and negative (12% vs 10.2% in positive- and negative-tested children, $P$ value = .48). Even after adjusting for age, race, and gender with

### Table 1: Demographics in association with COVID-19 test results in children evaluated at Rush University Pediatric COVID-19 Clinic between March and April 2019

| Characteristics | COVID-19–positive (%)<sup>a</sup> | COVID-19–negative (%)<sup>a</sup> | Odds ratio (95% CI)<sup>b</sup> |
|----------------|----------------------------------|---------------------------------|---------------------------------|
| Gender         |                                  |                                 |                                 |
| Male           | 15 (60%)                         | 240 (53.5%)                     |                                 |
| Race           |                                  |                                 |                                 |
| Non-Hispanic White |                              |                                 |                                 |
| Black          | 14 (56%)                         | 191 (42.5%)                     | 3.1 (1.23-5.34)                 |
| Hispanic       | 8 (32%)                          | 109 (24.3%)                     | 2.1 (0.97-4.67)                 |
| Asian          | 0 (0%)                           | 7 (1.6%)                        | n/a                             |
| Other          | 1 (4%)                           | 24 (5.3%)                       | 1.3 (0.56-4.34)                 |
| Age            | 9.72 (7.13)                      | 4.85 (5.65)                     | 1.09 (1.06-1.78)                |

<sup>a</sup>Odds ratio (95% CI) are calculated comparing children with positive vs those with negative COVID-19 tests by logistic regression adjusting for all demographics in one model.

<sup>b</sup>The % in each cell represents the percentage of cases with each characteristic (rows) in positive or negative groups, except the age which is represented by mean(SD).
logistic regression analysis, asthma was not associated with COVID-19 (adjusted P value = .47). Among admitted COVID-19–positive children, only one had asthma; however, he was admitted for a sickle cell acute pain crisis at the time of diagnosis of COVID-19. He did not require any additional bronchodilator treatment and was maintained on his home asthma regimen during admission.

As a secondary measure to confirm our results, we applied random stepwise matching among COVID-19–negative children to create a matched group for COVID-19–positive cases, first matching for gender and then for age (±1 year). Our large control group was sufficient to do a 2-to-1 match. Then, we compared the two age/gender-matched groups (COVID-19–negative and COVID-19–positive) for asthma and race. Similar to adjusted regression analysis, AA race was associated with higher odds of positive COVID-19, and asthma was not associated with differential COVID-19 risk. Given the low prevalence of COVID-19 in pediatric populations, demographic data and determination of risk factors in children are still emerging. Results from our study mirror previous observations regarding the overall low prevalence and milder presentation of COVID-19 in pediatric populations. Most children with COVID-19 exhibit mild symptoms and do not require hospitalization, and the majority of children recover from the illness in 1-2 weeks. In our study although only 20% of children needed hospitalization, 80% of the admitted children were AA. It is noteworthy that, while 25.1% of all tested children in our study were white, only 8% of COVID-19–positive cases were white. This disproportionately higher rate of COVID-19 in AA children and their higher rate of hospitalization is in line with the data in adult patients. Furthermore, in our series of children with COVID-19, all PICU-admitted children were AAs. This indicates that AA children are not only at higher risk of COVID-19 infection, but might also be at risk of a more severe infection course when they are hospitalized. This finding from our small number of cases, although not sufficient to draw any conclusion, calls for future larger studies to evaluate the course and outcome of COVID-19 in AA children. This increased risk of COVID-19 might be due to multiple factors. Individuals with underlying health conditions such as diabetes and obesity are at increased risk of COVID-19. Both obesity and diabetes are in rise and significantly more common in AA children than their white counterparts. Although these COVID-19 risk factors for COVID-19 are better defined in adults, they might impact the older children and adolescents as well. This is especially pertinent given our results that SARS-CoV-2 was more commonly detected in older children. Another explanation for the higher rate of COVID-19 in AA children might be due to inequities in healthcare access. Lack of access can result in delayed seeking of care in COVID-19–infected individuals and hence spread of the virus in their community. Suboptimal socioeconomic conditions evidenced by lower rates of homeownership in AA neighborhoods may be contributing to inability to fully shelter in place and higher exposure to COVID-19. These factors inevitably affect children as they frequently acquire infection through family members or other close contacts. Furthermore, the lower access to medical services and lower rates of preventive care visits can result in lack of awareness for preventive and cautionary practices in some populations.

The rate of asthma in our series was similar to the estimated prevalence of asthma in children in the state of Illinois. In our study of children, asthma was not associated with positive COVID-19 test even after adjusting for age, gender, and race. There were no admissions or ED presentations for asthma exacerbation among these positive COVID-19 patients. Thus, our study does not find asthma to be a risk factor for COVID-19 in children nor does a history of asthma portend a worse clinical course. This is consistent with the new findings, showing that allergic asthma is not a risk factor for COVID-19 and might even play a role as a protective factor. Small sample size was the main limitation of our study, likely also due to the overall low prevalence rate of COVID-19 in children; only 5.2% of children who were evaluated by our COVID-19 clinic for positive symptoms were positive. We acknowledge that our small number might have resulted in type 2 error to assess the risk of COVID-19 in asthmatic children, and larger population-based studies are needed to evaluate whether asthma is associated with differential risk of COVID-19 in children.

We found higher age, and AA race as risk factors of COVID-19 in children, which highlights the need for further attention and testing when appropriate in at-risk communities.

CONFLICT OF INTEREST
The authors have no conflict of interest to report.

AUTHOR CONTRIBUTION
Sindhura Bandi: Conceptualization (equal); Data curation (equal); Formal analysis (equal); Funding acquisition (equal); Investigation (equal); Methodology (equal); Project administration (equal); Resources (equal); Software (equal); Supervision (equal); Validation (equal); Visualization (equal); Writing-original draft (equal); Writing-review & editing (equal). Michael Zev Nevid: Conceptualization (equal); Data curation (equal); Formal analysis (equal); Funding acquisition (equal); Investigation (equal); Methodology (equal); Project administration (equal); Resources (equal); Software (equal); Supervision (equal); Validation (equal); Visualization (equal); Writing-original draft (equal); Writing-review & editing (equal). Mahboobeh Mahdavinia: Conceptualization (equal); Data curation (equal); Formal analysis (equal); Funding acquisition (equal); Investigation (equal); Methodology (equal); Project administration (equal); Resources (equal); Software (equal); Supervision (equal); Validation (equal); Visualization (equal); Writing-original draft (equal); Writing-review & editing (equal).

FUNDING INFORMATION
This study was conducted by internal departmental funding by Rush University. MM is supported by research grants from Brinson Foundation, NIH, and Medtronic.

KEYWORDS
asthma, COVID-19, pediatrics, race, risk factors
To the Editor,

The coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) appears milder in children, but little is known about neonates and about the chains of infections after delivery.1-3 When in early March 2020 a midwife in our large maternity and perinatal center returned from vacation in Ischgl, Austria, she triggered a COVID-19 outbreak affecting 36 midwives, nurses, and doctors. We reported previously on the successful containment of this outbreak and characterized the clinical symptoms and immunoglobulin development in staff members exposed to SARS-CoV-2.4,5

Here, we present the data of all deliveries with varying degrees of unprotected parental contact with SARS-CoV-2-infected personnel during the first, pre-containment, week of the outbreak. Of the 66 families concerned, 61 consented to a prospective study involving serial symptom interview, serial SARS-CoV-2 screening in throat-rinsing fluid (parents) and feces (infants), and serum IgA and IgG antibody studies (parents and infants) 4-5 weeks postpartum. Eighteen families had extensive unprotected contact with infected staff lasting >15 minutes at < 1.5 meters distance (Robert Koch Institute [RKI] risk category I). These families had their first SARS-CoV-2 test in the first week after delivery; they were quarantined for ≥2 weeks after discharge home and received weekly study visits. The remaining 43 less exposed families received only two visits.

We tested for SARS-CoV-2 by real-time reverse transcriptase-polymerase chain reaction (RT-PCR) for N2 and E gene (Xpert© Xpress SARS-CoV-2, Cepheid) and for serum IgA and IgG antibodies (EUROIMMUN AG) as previously published.5 In addition, to verify the antibody responses we performed a second antibody assay in postnatal SARS-CoV-2 infection and immunological reaction: A prospective family cohort study

DOI: 10.1111/pai.13302