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exclusion of patients who transferred to a different adult center, and missing outcome data.

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### An update from Minnesota on the seroprevalence of COVID-19 immunoglobulin G in our adult population

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**Background:** Since the onset of the COVID-19 global pandemic in 2019, much has been learned about the clinical course and epidemiology of SARS-CoV-2 in the cystic fibrosis (CF) population. Because of the severity of illness seen in this population during the H1N1 pandemic, people with CF were initially thought to be at greater risk of severe disease [1], but this community has shown significant resilience without clear evidence of more-severe COVID pneumonia [2]. The reasons for this are probably multifactorial, including younger age and long-standing infectious risk-mitigation practices such as social distancing and handwashing.

**Methods:** Individuals aged 12 and older who receive care at the Minnesota CF Center provided virtual consent and completed a brief online survey detailing possible exposures, symptoms of COVID-19, and behavioral data (e.g., handwashing, remote work opportunities, social-distancing practices). We extracted additional data from the electronic medical record to further risk stratify our patient cohort, including age, body mass index, sex, forced expiratory volume in 1 second, CF transmembrane conductance regulator modulator use, and diabetes. Participants were evaluated for COVID-19 immunoglobulin G (IgG) at the time of enrollment (0 months) and 6 and 12 months after enrollment.

**Results:** Data were obtained 120 enrollees with an average age of 37; 50% were female. Preliminary data show that 25 (20.1%) of those tested had evidence of a natural COVID infection (IgG+ pre-vaccination or nucleocapsid Ab+). Two of these were hospitalized with COVID pneumonia between December 2020 and October 2021. Induced IgG ranged between 0 and 12 months' duration. At this time, 85% of the enrolled participants have been vaccinated against SARS-CoV-2: 67% with Pfizer-BioNTech, 25% with Moderna, and 9% with Johnson & Johnson. This is significantly higher than the vaccination rate of the general population in Minnesota, currently 66%.

**Conclusions:** As SARS-CoV-2 evolves, so too must our understanding of its natural history in people with CF. Our study shows that induced immunity through vaccination results in prolonged (12 months) IgG production in a subset of patients. The higher vaccination rate in the CF population along with infection risk-reduction practices have all helped reduce disease severity from COVID-19.

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#### References

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### Association between *Pseudomonas aeruginosa* infection stage and lung function trajectory in children with cystic fibrosis

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**Background:** The effect of chronic *Pseudomonas aeruginosa* infection on lung function trajectory in people with cystic fibrosis (CF) is not known. We aimed to evaluate the association between longitudinal lung function (percentage predicted forced expiratory volume in 1 second (FEV<sub>1</sub>pp) with different stages of *P. aeruginosa* infection (never, incident, chronic) using four definitions of chronic *P. aeruginosa* in a U.S. national cohort of children with CF.

**Methods:** Participants in the Early *Pseudomonas* Infection Control Observational Study diagnosed with CF before age 3 contributed encounter-based data through the U.S. CF Patient Registry 1992–2017. Follow-up was censored at the time of solid organ transplant or gaps of more than 2 years in culture or FEV<sub>1</sub> data. Cubic spline linear mixed-effects models were used to evaluate the association between *P. aeruginosa* stage and longitudinal FEV<sub>1</sub>pp. Participants contributed to the *P. aeruginosa* never FEV<sub>1</sub> curve until they acquired incident *P. aeruginosa*, at which point they began contributing to the incident *P. aeruginosa* FEV<sub>1</sub> curve. If they developed chronic *P. aeruginosa*, they then began contributing to the chronic *P. aeruginosa* FEV<sub>1</sub> curve. Models contained interaction terms between age and *P. aeruginosa* stage (never, incident, chronic) and were adjusted for birth cohort, diagnosis after a positive newborn screen, sex at birth, race, ethnicity, insurance at first FEV<sub>1</sub>, *CFTR* genotype (minimal vs. residual function), and the following time-varying covariates: CF-related diabetes, modulator use (ivacaftor, lumacaftor/ivacaftor), and respiratory culture positive for other individual CF pathogens. We used four chronic *P. aeruginosa* definitions [1]: 2 *P. aeruginosa*+ years over 3 years, 3 *P. aeruginosa*+ years over 4 years, 3 *P. aeruginosa*+ age-quarters over 2 years, 2 *P. aeruginosa*+ age-quarters over 1 year.

**Results:** Of 1,264 subjects born between 1992 and 2006 that provided a median 9.5 years (interquartile range 0.25–15.75 years) of follow-up, 89% developed incident *P. aeruginosa*, and 39% to 58% developed chronic *P. aeruginosa* depending on the definition. Model estimates were similar for all chronic *P. aeruginosa* definitions, so we show results using the second definition (3 *P. aeruginosa*+ years over 4 years) in Figure 1. The top panel shows the model estimates of FEV<sub>1</sub> (mean and 95% confidence intervals) according to age and *P. aeruginosa* stage. The bottom panel shows the number of FEV<sub>1</sub> values available at each age for each *P. aeruginosa* stage. The estimated mean FEV<sub>1</sub>pp is lower after initial *P. aeruginosa* acquisition and lower still after chronic *P. aeruginosa* infection at all ages; this effect increases with age. Similarly, the slope of FEV<sub>1</sub>pp decline is greater in those with chronic *P. aeruginosa*, particularly after age 12.