issues noted in this cohort were lack of compliance, varying levels of engagement and missed clinic appointments. When the pandemic was declared, there were concerns of delayed presentations due to fears of contacting Covid-19. Towards addressing this, virtual asthma clinics were started, running seven days a week to support children with asthma and their families. **Objectives** The objective was to learn from the pandemic dataset and take the lessons moving forward.

Methods We analysed the data of PED attendances and admissions with asthma/VIW in 2020, extracted from the electronic database and compared it with similar data from 2019.

Results There was an unprecedented reduction in PED attendances for children with asthma and VIW from the time of the first lockdown in March 2020 to August 2020 (45% reduction in March 2020 compared to the previous year, 91% reduction from April to July 2020 compared to the previous year). There was a 80 -90% reduction in admissions for asthma/VIW during this time period and fewer severe asthma presentations. Factors like lockdown, closure of schools and hand washing were probably the main contributors in interrupting the chain of transmission of not only COVID-19 but other viruses too. Anecdotal evidence from clinicians who led the virtual asthma clinics reported improved engagement and compliance with preventers especially from repeat attenders and teenagers who previously had shown poor compliance. Change in patient and parent specific behaviour due to fears of contracting COVID-19 contributed to better self-management of paediatric asthma.

However a seasonal spike in asthma attacks which is common in September (the 'September epidemic') did happen in 2020 as children returned to school. There was a 45 - 55% reduction in attendances in October and November 2020. With the announcement of the second lockdown in December asthma/VIW attendances dropped by 84–91% from December to February 2020, a dramatic decline. Prevalence of COVID - 19 or endemic winter viruses did not seem to contribute to attendances or admissions for children with asthma and VIW.

Conclusions This COVID-19 pandemic has shown us a snapshot of how childhood asthma can be managed in a different way. Identifying the patterns of change is important for the future as this experience can be used to promote integrated care for children with asthma and VIW. The emerging concept of telemedicine has also contributed significantly. An existing strategy of engaging children and families who present to PED with an acute exacerbation of asthma to raise education and awareness was already present in this trust since 2019. Learning from the pandemic experience, we think that media exposure and health care pathways could provide the right information and guidance to families of children with asthma/VIW.

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RESIDENT MEMORY AIRWAY T CELLS PROTECT AGAINST RSV INFECTION IN THE ABSENCE OF ANTIBODY

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Background Tissue resident memory T (Trm) cells act as sentinels and early responders to infection. (RSV)-specific Trm cells have been detected in the lungs after human RSV infection, but their role is unknown. To dissect the protective function of Trm cells, mice were infected with RSV; infected mice developed antigen-specific CD8⁺ Trm cells in the lungs and airways. Intranasally transferring cells from the airways of previously infected animals to naïve animals reduced weight loss on infection in the recipient mice. Transfer of airway CD8 cells led to reduced disease and viral load in recipient mice. Because DNA vaccines induce a systemic T-cell response, we compared vaccination with infection. Intramuscular DNA immunization induced RSV-specific CD8 T cells, but they were not protective. Infection but not immunization induced antigen-specific Trm cells in a range of tissues. These findings demonstrate a protective role for airway CD8 against RSV and support the need for vaccines to induce antigen-specific airway cells.

Objectives In this study the role of airway resident memory T cells (Trm) in protection against RSV infection was examined.

It was hypothesised that since Trms are present at the site of infection and can act as sentinels, their induction would lead to rapid pathogen clearance.

Methods Antigen specific Trm cells (CD69+CD103+) were characterized in the lungs and other tissues of mice after infection or vaccination.

For vaccination, DNA was delivered intramuscularly in a prime-boost-boost regimen separated by two weeks.

To dissect the protective function of Trm, cells were obtained by bronchoalveolar lavage from RSV experienced mice, sorted and transferred intranasally into naïve animals prior to intranasal challenge a day later with RSV.

Results 1. Prior RSV infection protects against subsequent infection and induces both RSV specific antibody and CD8 T cells. Naïve or previously RSV infected mice were infected with RSV intranasally, weight was measured daily. Viral load was measured on day 4 after infection. Anti-RSV antibody responses were measured on day 7 after infection.

2. Airway cells, not splenocytes from RSV experienced mice can protect from disease in the absence of antibody. Mice received cells from airways and spleens of RSV exposed or PBS exposed mice intranasally prior to infection with RSV. Viral load was measured on day 7.

3. Immunisation and infection induce different antigen specific T cell populations in different tissues. Mice were either vaccinated with RSV DNA vaccine or intranasally infected with RSV. After 8 weeks all mice were sacrificed. The left and right flank skin and muscle, blood, spleen, airway and lung cells were analysed. RSV specific CD8⁺ T cells and proportion of Trm were assessed.

Conclusions RSV infection leads to CD8⁺ Trm formation in the lungs and the airways of mice.

Intra-airway cell transfer of RSV experienced CD4 or CD8 cells to naïve animals protects from disease upon challenge in the absence of antibodies.

RSV infection leads to dissemination of RSV specific CD8⁺ Trm cells however this is not reproduced by IM vaccination.

Localised delivery of vaccines to the lungs may be needed to generate formation of Trm in the airways.