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004 SARS-CoV-2 entry factors are expressed in nasal, ocular, and oral tissues: implications for COVID-19 prophylaxes/therapeutics

Ivan Lee[1], Tsuguhisa Nakayama[1], Sizun Jiang[1], Yury Goltsev[1], Christian Schürch[1], Bokai Zhu[1], David McIlwain[1], Pauline Chu[1], Han Chen[1], Alexandar Tzankov[2], Matthias Matter[2], Jayakar Nayak[1], Garry Nolan[1]; [1]Stanford University School of Medicine, [2]Institute of Medical Genetics and Pathology, University of Basel, Switzerland.

RATIONALE: Tissue tropism is one key to understanding the pathogenesis of SARS-CoV-2, the causative agent of the ongoing severe acute respiratory disease pandemic COVID-19. We characterized the protein expression of the essential SARS-CoV-2 receptor, angiotensin-converting enzyme 2 (ACE2), and the critical SARS-CoV-2 entry factor, transmembrane protease, serine 2 (TMPRSS2), in human aerodigestive and ocular tissues to gain insights into initial SARS-CoV-2-host interactions.

METHODS: Immunofluorescent staining was simultaneously performed on tissue microarrays consisting of normal human head & neck tissues. Tissues from SARS-CoV-2-infected patients were collected during autopsy. Imaging was performed using confocal microscopy, and quantification of fluorescent intensity was done using a custom open-source software in ImageJ.

RESULTS: ACE2 and TMPRSS2 protein expression localize to a variety of human airway, ocular, and oral epithelial surfaces, suggesting that SARS-CoV-2 has the capability to enter through all mucosal surfaces of the face. Notably, ACE2 and TMPRSS2 are both very highly expressed in the motile cilia of the nasal mucosa. Finally, *SARS-CoV-2 Spike* transcripts are readily detected in the nasal epithelia of SARS-CoV-2-infected patients.

CONCLUSIONS: ACE2 & TMPRSS2 are expressed on nasal, ocular, and oral epithelial surfaces, and are notably present within the motile cilia of the airway. As breathing occurs primarily through the nasal passage, the dense motile cilia in the nasal epithelia are predicted to readily encounter SARS-CoV-2 during viral transmission and serve as a predominant initial/early site of infection. Prophylaxes and therapeutics for COVID-19 should, therefore, focus on a nasal route of administration. Similarly, proper eye protection should be worn during certain circumstances

005 Picornavirus Infection of Esophageal Epithelial Cells

Vinay Goswamy[1], Paul Fichtinger, BS[2], Elizabeth McKernan, BS[1], Sameer Mathur, MD PhD FAAAAI[3]; [1]University of Wisconsin - Madison, [2]University of Wisconsin, [3]University of Wisconsin School of Medici.

RATIONALE: Eosinophilic esophagitis (EoE) is a disorder characterized by inflammation and fibrosis in the esophageal tissue. There are mechanistic parallels between EoE and other Th2 inflammatory conditions including asthma. One of the contributors to inflammation in asthma is viral infection. We hypothesized that viral infection of esophageal epithelium may also contribute to inflammation in EoE.

METHODS: We cultured immortalized esophageal epithelial cells (EPC2-hTERT) as a monolayer with Interleukin-13 (IL-13) to replicate the esophageal environment in EoE. Picornaviruses (Rhinovirus 1A, Rhinovirus 16, and Enterovirus D68) were used to infect EPC2 cultures for 48 hours. RNA samples were collected for analysis of viral replication and for gene expression from the EPC2 cells using PCR. We measured Rhinovirus and Enterovirus RNA levels (for virus replication) and IFIT1 gene expression (for anti-viral response) by PCR.

RESULTS: We confirmed mRNA expression of cell surface receptors for picornaviruses, specifically LDLR, ICAM-1, and ICAM-5, which are the receptors for RV1A, RV16, and Enterovirus-D68, respectively. mRNA expression of LDLR was 4 log higher than ICAM-1. Rhinovirus 1A replication was present with monolayer cultures of EPC2 cells with IL-13 ($n=4$, $p<0.001$). Increased gene expression of IFIT1 confirmed anti-viral responses in the monolayer cultures of EPC2 cells ($n=4$, $p<0.001$).

CONCLUSIONS: In the EPC2 cell line in vitro model of esophageal epithelium, we demonstrate expression of picornavirus receptors (LDLR, ICAM-1, and ICAM-5), replication of RV1A, and increased expression of a virus and interferon-related gene (IFIT1). These findings provide evidence that a viral process may be contributing to the inflammation associated with EoE in some individuals.

006 Food Reintroduction after Passing an Oral Food Challenge: A Cross-Sectional Structured Interview-Based Assessment of Barriers, Challenges, and Impact on Quality of Life

Christina Ditlof[1], Roxanne Hummel[1], Samantha Wong, MSc, RD[1], Mara Alexanian-Farr, MSc, RD[1], Slavka Zahrebelny[1], Jennifer Hoang[1], Lisa Hung[1], Julia Upton, MD[1], A. D. E. L. L. E. ATKINSON, MD, FRCP[1], Maria Asper, MD[1], David Hummel, MD[1], Thomas Eiwegger, MD[1]; [1]Hospital for Sick Children.

RATIONALE: Asymptomatic sensitization and equivocal history often results in long periods of avoidance. In the case of a negative OFC (OFC^{neg}), the patient must reintroduce the previously avoided food on a regular basis. This population allows for the evaluation of the impact of food allergy delabelling and investigation of challenges linked to food introduction.

METHODS: The validated Food Allergy Quality of Life Questionnaire – Parent Form, Child Form, and Teenager Form (FAQLQ-PF, CF, TF), and motivators and barriers for food reintroduction were assessed in a Canadian cohort of 96 parents, children and adolescents with an OFC^{neg} via a structured interview. Using the FAQLQs, quality of life (QoL) was compared to a matched cohort of food allergic pediatric patients and caregivers ($n=90$).

RESULTS: Overall QoL in the OFC^{neg} cohort was significantly better in parents (1.92 ± 0.19 vs 3.02 ± 0.18, $p<0.0001$) and children (2.65 ± 0.25 vs 3.93 ± 0.25, $p=0.001$) but not adolescents (3.16 ± 0.31 vs 3.91 ± 0.23) compared to the matched food allergic cohort. This effect was independent of the type and amount of food consumed. Information given by clinicians post-OFC was a primary motivator for food reintroduction. The most important barriers to reintroduce the culprit food were fear of a reaction or dislike of the food.

CONCLUSIONS: Parents and children benefit from food allergy delabelling demonstrated through improvement in QoL irrespective of the amount of food reintroduced. The information given by clinicians after a negative OFC to promote successful food reintroduction is of clinical importance.