

**Ziegler et al., Cell: SARS-CoV-2  
receptor ACE2 is an interferon-  
stimulated gene in human airway  
epithelial cells and is detected in  
specific cell subsets across tissues**

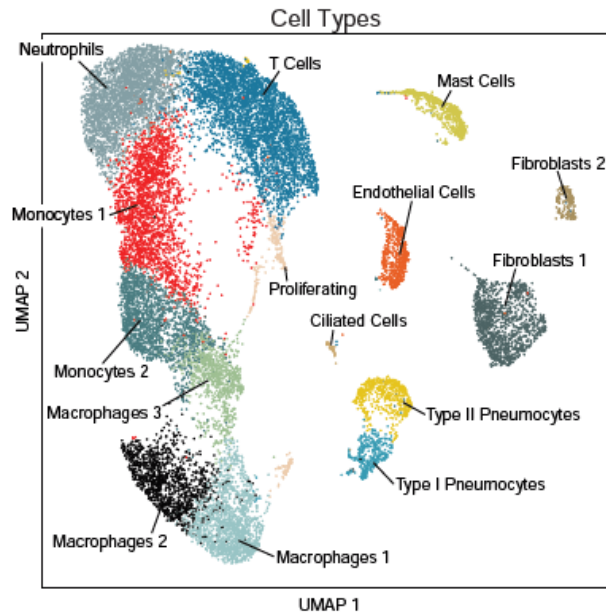
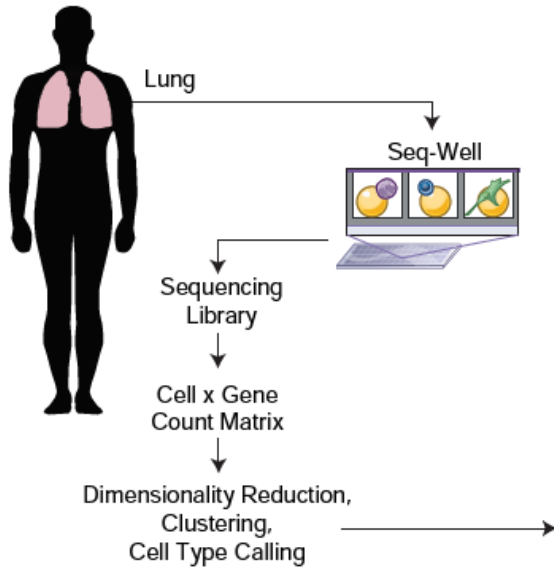
# ACE2 und TMPRSS2

- angiotensin converting enzyme 2 (ACE2) is the receptor for SARS-CoV
- SARS-CoV-2 spike (S) protein binds ACE2 on host cells with significantly higher affinity than SARS-CoV-S
- host protease Type II transmembrane serine protease TMPRSS2 mediates S protein activation and viral entry
- ACE2 expression in epithelia of human lung and small intestine, which subset?
- Ziel 1: Identification of cell subsets targeted by SARS-CoV-2 (ACE2+) and (ACE2+/TMPRSS2+)

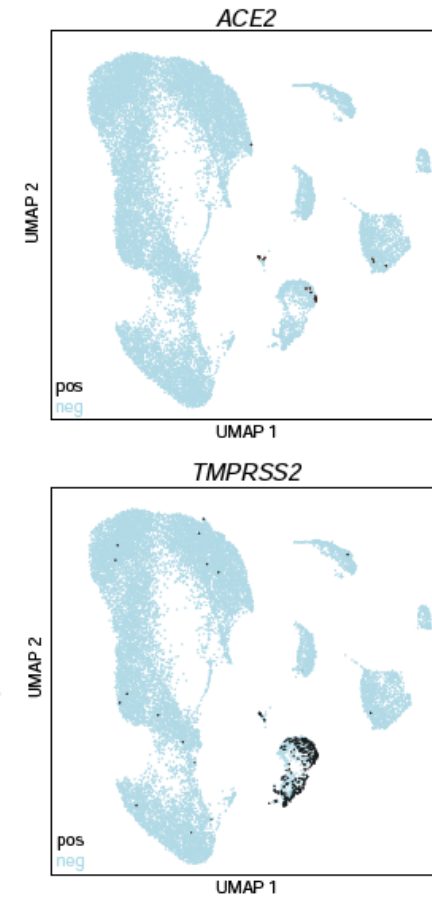
# Was wurde gemacht?

- Analyse von **single-cell RNA-sequencing data sets** from non-SARS-CoV-2 infected samples (human, HIV, Tb Patienten) tissues clinically identified to harbor virus in COVID-19
- Welche Zellen exprimieren ACE2 und TMPRSS2?
- **Lung Epithelial Cell Expression of Host Factors Used by SARS-CoV-2 in Humans**
- **Upper Airway Expression of Host Factors used by SARS-CoV-2**

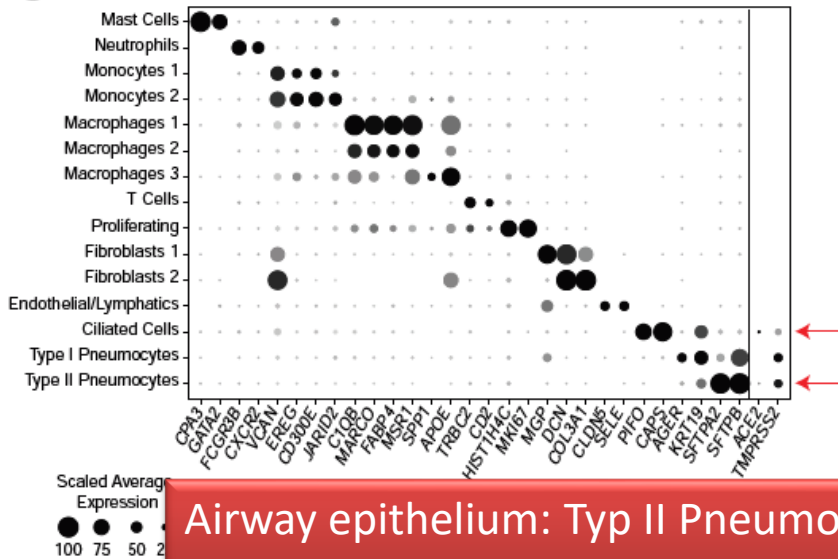
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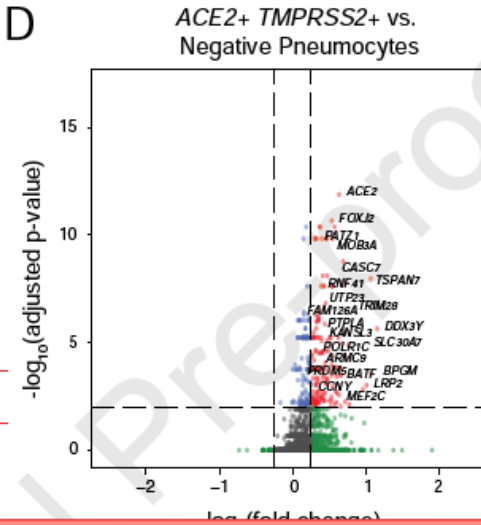
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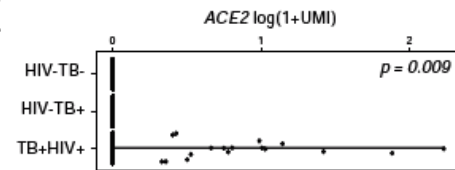
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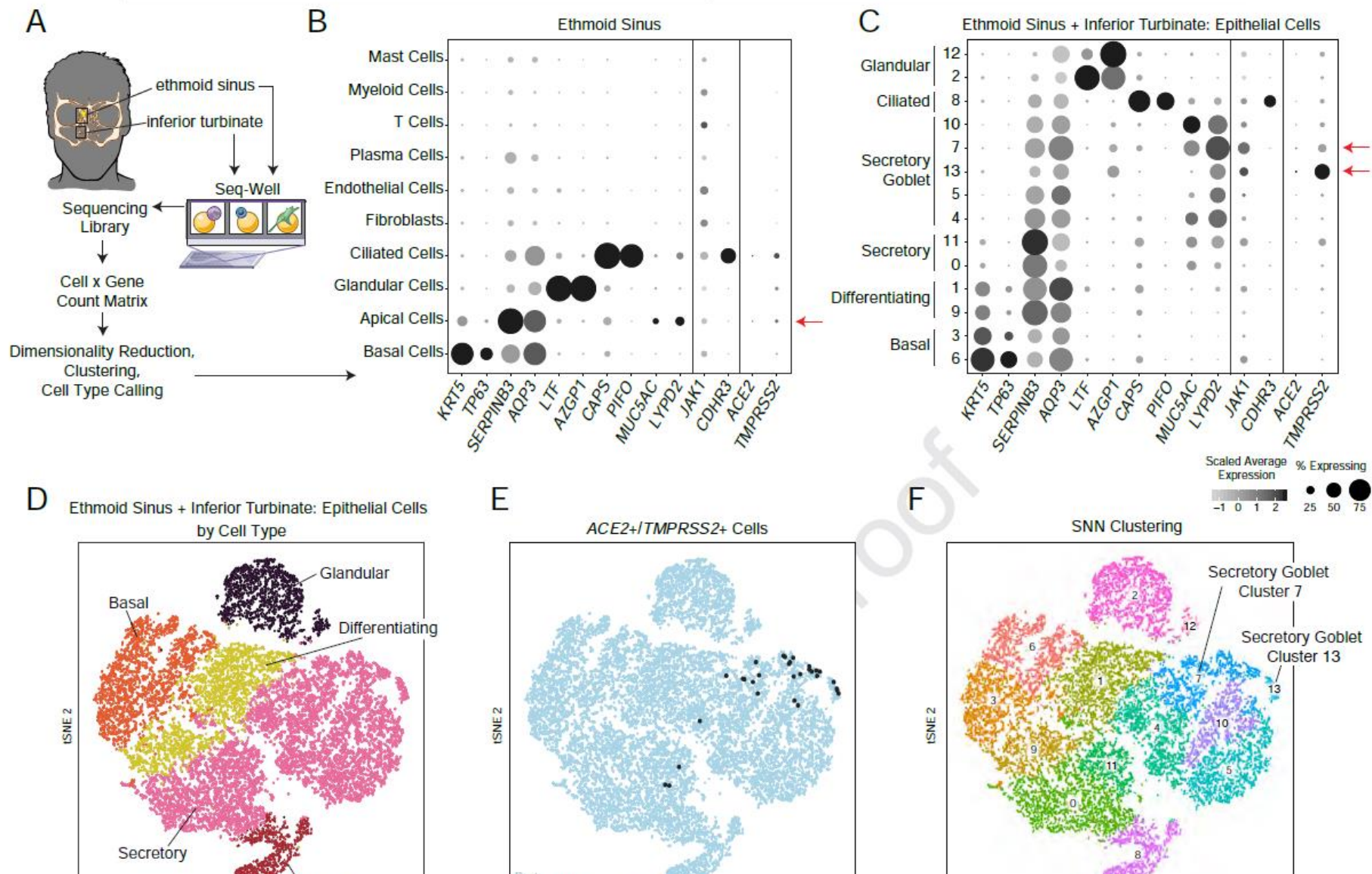


E



Airway epithelium: Typ II Pneumozyten (0,8%) und Zilienzellen (5%) sind ++ (prone for SARS-CoV2 infection)

Figure 4



Nasal mucosa: 1,6% der sekretorischen Becherzellen exprimieren ACE2 und TMPRSS2

# Ergebnis:

- *ACE2* and *TMPRSS2* co-expressing cells wurden im humanem Lungenepithel, im Ileum und in der Nasenschleimhaut gefunden
- könnten ein Target für SARS-CoV2 darstellen
- In diesen Zellen wurde significant upregulation of IFN-stimulated genes observed, potentially indicative of association between *ACE2* expression and components of the IFN-signaling pathway
- human primary basal were cultured and treated with IFNa2, IFNg, IL-4, IL-13, IL-17A, or IL-1b
- Only IFNa2 and IFNg led to upregulation of *ACE2* Type I Interferon
- IFNa Drives *ACE2* Expression in Primary Human Nasal Epithelial Cells

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