



## Diagnosis and Management of Bronchial Epithelial Cells

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### Abstract

Pulmonary lesions were found to be completely absorbed in 53.0% of patients during the 3rd week after discharge, implying that pulmonary damage caused by COVID-19 could be potentially repaired without any sequel.

**Keywords:** Ventilator surface; Lung parenchyma; Bronchiolar narrowing; Respiratory failure; Inflammatory stenosis; Emphysema;

### Introduction

However, more than 40% of patients demonstrated residual abnormalities, including ground-glass opacity and fibrous stripe as the main CT manifestations at the 3-week radiological follow-up, for whom further radiological follow-up was continued. Two weeks was the appropriate median resolution period in this cohort. The CT score of non-ground-glass opacity lesions was used to evaluate residual pulmonary involvement. This was because an extended ground-glass opacity area with decreased density may have occurred in some patients after discharge, and ground-glass opacity is even a basic manifestation of convalescence, which could have led to over-estimation of the CT scores. As demonstrated in this study, patients with a CT score  $\leq 1$  at discharge did not show a faster resolution than patients with a CT score  $> 1$ , which indicated a stable baseline in the CT scores of all patients at discharge. Previous studies have reported that older age and male sex are high risk factors for worse outcomes in patients with COVID-19. However, in this study, under the milieu of similar residual pulmonary lesions in the patients at discharge, females did not show a faster resolution rate [1]. On the other hand, younger age was associated with a better outcome in the convalescence period in this study, which is consistent with a former study. Radiological abnormalities have been reported to start resolving in the late phase in SARS, and the typical later-stage CT appearances were a coarse reticular pattern and ground-glass opacity in the anterior part of the lungs [2].

### Discussion

For SARS, intra-lobular and interlobular septal thickening was observed to predominate over ground-glass opacity even at 84 months. In our cohort, ground-glass opacity and fibrous stripe were the main imaging findings during the convalescence of COVID-19 pneumonia, which could be gradually absorbed completely, while a crazy-paving pattern was not demonstrated [3]. This observation addressed the question raised by Shi H et al. about whether the fibrosis in COVID-19 is irreversible. There were three patterns in the residual lesions with proper evolution after discharge, the extent of ground-glass opacity was reduced and gradually faded until it completely disappeared; fibrous stripes developed within the ground-glass opacity area, followed by gradual resorption and disappearance; and fibrous stripes gradually reduced with decreasing density [4]. Tinted sign and bronchio-vascular bundle distortion as two special features were discovered during the evolution. The tinted sign was demonstrated as extension of the ground-glass opacity area and a decrease in density, which might follow the melting sugar sign. In this study, patients were found with a tinted sign. The appearance of this pattern probably implies the gradual resolution of inflammation with re-expansion of alveoli and hence the resolution or recovery of illness, for which the pathological evidence merits

further investigation. In the mixed pattern, patients were found to have bronchio-vascular bundle distortion, with 4 patients with complete resolution during 17–37 days after discharge [5]. This may be caused by inflammatory distraction or sub-segmental atelectasis. In addition, patients had varying extents of residual fibrous stripes, and the lesions were not fully resorbed at the end of the observation, which may be attributed to the limited follow-up time. The other CT manifestations in convalescence, such as thickening of the adjacent pleura and small pleural effusion, could also be absorbed completely. A main limitation of the current study is that no critical patients were involved in this study, as they were still hospitalized in our centre at the end of the study. Second, only semi-quantitative estimation was carried out in this study for the complicated radiological characteristics in the convalescent stage. The dynamic resolution process of lung lesions in discharged patients recovering from COVID-19. For most of the discharged patients, two weeks after discharge might be the optimal time point for early radiological estimation. Elderly patients need a longer time to reach complete radiological resolution. This study may help to understand the recovery course of this disease and indicate an optimized time point for chest CT scans in discharged patients [6]. The contribution of exposure to cigarette smoke to the development and progression of chronic obstructive pulmonary disease, cancer, and cardiovascular diseases is widely recognized. Electronic cigarettes are commercially available since 2004, but patents for similar devices reach back to 1965. Electronic cigarettes are devices that produce a vapour by heating a liquid that usually contains a mixture of glycerol, propylene-glycol, water, flavours, and different concentrations of nicotine. The flavours cover a broad range of tastes from fruit or spices, to different brands of tobacco. An intense scientific and political discussion about the toxicity and potential harm reduction of Electronic cigarettes is ongoing. Glycerol-propylene-glycol-water mixtures have been in use for a long time as artificial fog in aviation emergency training and entertainment business, but only a few studies about possible side effects exist before the emergence of Electronic cigarettes. Several studies have analysed the effects of ECIGs on lung cells with the goal to evaluate toxic effects on cells and tissues. A number of negative outcomes on different tissue culture systems have been reported. In parallel, several studies compared ECIGs with TCIGs and often found

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decreased acute toxicity. Electronic cigarettes -vapor induced DNA-strand breaks in vitro that are known to be induced by oxidative modifications of DNA by free radicals. Several studies showed increased cytotoxicity and oxidative stress of flavored Electronic cigarettes -vapour in vitro and in vivo [7]. Additionally, it has been shown that Electronic cigarettes vapour may damage the pulmonary endothelial barrier and induce pulmonary neutrophilic inflammation. A recent study showed differences in gene expression in differentiated bronchial epithelial cells between TCIG- and Electronic cigarettes -exposed cells, with and without nicotine, and showed differences in gene expression signatures in various pathways like phospholipid and fatty acid triacylglycerol, which were significantly enriched after ECIG-exposure. In the present study we aimed to compare the acute effects of Electronic cigarettes -vapor and TCIG-smoke on inflammation, host defence and cellular activation of human airway epithelial cells. We used an in vitro TCIG-exposure model and adopted it to the vaporization of Electronic cigarettes-liquid. Although we are aware that the composition of TCIG-smoke, besides nicotine and glycerol, is highly different from Electronic cigarettes -vapour, we normalized the amount of Electronic cigarettes -vapour to the content of nicotine from our established TCIG-exposure model [8]. This has not been done in previously published studies and was important for direct comparison of the effects of Electronic cigarettes -vapor and TCIG-smoke exposure. We chose nicotine consumption as a normalization factor to account for the needs of smokers to meet their demands for nicotine, when switching between TCIGs and ECIGs. The human lung adenocarcinoma cell line Calu-3 was cultured in DMEM/F12 and the human bronchial epithelial cell line NCI-H292 was cultured in RPMI medium both supplemented with 10% foetal bovine serum and 1% penicillin-streptomycin. Primary human bronchial epithelial cells were isolated from large airways from samples obtained from macroscopically healthy areas of resected lung samples during surgery as described before. The primary cells used within this study were from 3 different donors of Caucasian origin to account for intra-individual differences. The isolation and use of human specimen was approved by the ethics committee of the Landesärztekammer des Saarlandes [9]. The primary cells were grown in airway epithelial cell growth medium with growth supplement and 1% penicillin-streptomycin, passaged once and frozen for later use. The experiments were repeated three times all cells were tested in regular intervals and found to be mycoplasma-free. To analyse the epithelial barrier integrity, the translocation of dextran from the apical to the basolateral side of the cell layer was determined as described before. Directly after exposures, 200 µL of 10 mg/mL fluorescein labeled isothiocyanate-dextran in PBS or PBS containing heat inactivated *P. aeruginosa* PAO1 were applied directly on cells. 100 µL of the basolateral solution was collected after 24 hours and the fluorescence was measured. The fluorescence intensity was calculated against a standard curve of known concentrations of FITC-Dextran. We determined the IL-8 concentration in the basolateral solution of the air-liquid interface before and after exposure, by enzyme-linked immune-sorbent assay according to the manufacturer's instructions. We used a TECAN Ultra

384 ELISA reader and the software Magellan [10]. Total RNA of cells was isolated by Nucleospin RNA Kit according to manufacturer's instructions and used for whole-genome gene expression array analysis or validation of the array by qRT-PCR. cDNA was synthesized with the Revert Aid First strand cDNA Synthesis Kit and oligo dT18-primers. The expression of the different genes was quantified by using the SensiMix SYBR & Fluorescein Kit and the C1000 Touch Thermal Cycler. The specific primer sequences used in this work can be found in the supplementary part. The expression was quantified by the  $\Delta\Delta$ -method. Primary human bronchial epithelial cells from different donors were mixed and cultured as described above.

## Conclusion

After differentiation on transwell plates, the cells were exposed to TCIG and ECIG. Twenty-four hours after exposure RNA was isolated according to the manufacturer's instructions. The RNA was used for expression analysis on an Illumina HumanHT-12 v4 Expression Bead Chip according to the manufacturer's instructions. The analysis was performed by the Institute of Clinical Molecular Biology

## Acknowledgement

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## Conflict of Interest

None

## References

1. Cooper GS, Parks CG (2004) Occupational and environmental exposures as risk factors for systemic lupus erythematosus. *Curr Rheumatol Rep* EU 6:367-374.
2. Parks CG, Santos ASE, Barbhaiya M, Costenbader KH (2017) Understanding the role of environmental factors in the development of systemic lupus erythematosus. *Best Pract Res Clin Rheumatol* EU 31:306-320.
3. M Barbhaiya, KH Costenbader (2016) Environmental exposures and the development of systemic lupus erythematosus. *Curr Opin Rheumatol* US 28:497-505.
4. Gergianaki I, Bortoluzzi A, Bertias G (2018) Update on the epidemiology, risk factors, and disease outcomes of systemic lupus erythematosus. *Best Pract Res Clin Rheumatol* EU 32:188-205.
5. Cunningham AA, Daszak P, Wood JLN (2017) One Health, emerging infectious diseases and wildlife: two decades of progress? *Phil Trans UK* 372:1-8.
6. Sue LJ (2004) Zoonotic poxvirus infections in humans. *Curr Opin Infect Dis* MN 17:81-90.
7. Pisarski K (2019) The global burden of disease of zoonotic parasitic diseases: top 5 contenders for priority consideration. *Trop Med Infect Dis* EU 4:1-44.
8. Kahn LH (2006) Confronting zoonoses, linking human and veterinary medicine. *Emerg Infect Dis* US 12:556-561.
9. Slifko TR, Smith HV, Rose JB (2000) Emerging parasite zoonosis associated with water and food. *Int J Parasitol* EU 30:1379-1393.
10. Bidaisee S, Macpherson CNL (2014) Zoonoses and one health: a review of the literature. *J Parasitol* 2014:1-8.