Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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CoV-2 infection during the same period 4.3% (95%CI; 3.6-5.2) p<0.001. Among 653 patients receiving active cancer therapy during this period, 24 (3.7%) developed COVID-19 and required admission, 4.2% of whom were receiving chemotherapy, 9.5% immunotherapy and 2.1% targeted therapies. Lung and breast cancer were the most frequent (26.1%), followed by colorectal (19.6%) and breast cancer. No significant differences due to the cancer treatment received were observed. Mortality in lung cancer patients was the highest (25%). The univariate analysis (between p who developed serious event vs. those who did not), showed that higher Brescia, CURB-65 scale, lactate dehydrogenase (LDH) or C-reactive protein (CRP) levels at admission, the greater risk of developing severe complications (p<0.05).

Conclusions: Patients with cancer, especially lung cancer, and SARS-CoV2 infection have a worse overall prognosis than the general population. Objective parameters such as LDH, CRP at admission, Brescia index or CURB-65 should alert us to a more serious evolution and suggest early an early intensive care unit (ICU) admission.

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**Table: 1734P Univariate analysis of risk factors for mortality in COVID-19**

| Variable                  | Alive | Dead | p-value |
|---------------------------|-------|------|---------|
| Age (years)               |       |      |         |
| South Asian ethnicity *   | 16 / 8 | 20 / 17 | 0.03   |
| Cardiovascular disease *  | 41 / 20 | 36 / 31 | 0.04   |
| Cerebrovascular disease   | 23 / 11 | 26 / 22 | 0.02   |
| Chronic kidney disease *  | 17 / 8  | 21 / 18 | 0.02   |
| Hypertension *            | 92 / 46 | 68 / 58 | <0.05  |

* shown as n / %, ^ shown as median (IQR)

Conclusions: Along with known risk factors, cancer confers an independent risk for mortality in COVID-19. Taken together, our findings support the need to continue ‘shielding’ patients with cancer from exposure to COVID-19 infection. Increasing age and co-morbidity should be taken into account when assessing risk for severe COVID-19 infection in cancer patients.

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**1735P SARS-CoV-2 infection induces EMT-like molecular changes, including ZEB1-mediated repression of the viral receptor ACE2, in lung cancer models**

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Background: SARS-CoV-2 infection is the cause of the respiratory illness COVID-19, which presents most frequently with respiratory symptoms. SARS-CoV-2 cell entry requires interactions with ACE2 and TMPRSS2 on the surface of the host cell. Cancer patients and, specifically, those with thoracic malignancies seem to experience poorer clinical outcomes.

Methods: We utilized bulk and single-cell transcriptional data from a combination of normal and malignant tissues and cells from aerodigestive and respiratory tracts to explore mechanisms governing the expression of ACE2 and TMPRSS2. Additionally, we determined the effect of EMT induction, ZEB1 modulation, and SARS-CoV-2 infection on ACE2 expression.

Results: Our bulk data suggests that aerodigestive and lung cancer models express a broad range of ACE2 and TMPRSS2, particularly in epithelial cells, and would serve as good models for studying SARS-CoV-2 infection. We assessed the relationship between ACE2 and epithelial differentiations in numerous datasets, and found consistent positive correlations with transcriptional and microRNA signatures of epithelial differentiation. The miR-200 family — zinc finger E-box-binding homeobox 1 (ZEB1) pathway, which is an established regulator of EMT, also directly regulates ACE2 expression, likely via putative ZEB1 repressor sites located in the ACE2 promoter. Furthermore, SARS-CoV-2 infection reduces ACE2 expression and shifts cells to a more mesenchymal phenotype with loss of EPCAM and upregulation of ZEB1 and other EMT-associated genes.

Conclusions: ACE2-positive cells are almost exclusively epithelial and unexpectedly rare, considering the devastating impact of this infection. Following viral entry, SARS-CoV-2 infection induces molecular changes within the cells that are reminiscent of EMT, including increased ZEB1. ZEB1, in turn, appears to directly repress the expression of ACE2. This SARS-CoV-2-induced ACE2 deficiency, compounded by the downregulation of genes, including claudins, which play a critical role in restricting epithelial and endothelial permeability, exposes respiratory cells to increased risk of edema and acute respiratory distress syndrome (ARDS).

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**1736P Elevated AXL expression following SARS-CoV-2 infection in non-small cell lung cancer**

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Background: Patients with thoracic cancers affected by the coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), tend to have poor clinical outcomes. AXL, a TAM (Tyro3, AXL, Mer) family receptor tyrosine kinase, is a known mediator of epithelial to mesenchymal transition (EMT) and therapeutic resistance in non-small cell lung cancer (NSCLC) and other cancers. Additionally, AXL plays a role in efficient Ebola and Zika viral entry and...