The impact of air pollution on SARS-CoV-2 infection of olfactory cells
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Overview

Air pollution is believed to be a major factor in 15% of all COVID-19 deaths. Neurological changes accompany at least 30% of COVID-19 cases. The olfactory epithelium provides a natural bridge between the external environment and the brain. ACE2, the main SARS-CoV-2 cell receptor, is expressed in human olfactory cells.

We will investigate the impact of air pollution on SARS-CoV-2 infection in different age and health cohorts such as dementia.

Aims

- To investigate infectivity and cell toxicity of SARS-CoV-2 in human olfactory cells.
- To evaluate the impact of SARS-CoV-2 on olfactory cells from different cohorts with varying ACE2 expression (age, sex, and different neurological or other underlying disorders).
- To determine the effect of air pollution on SARS-CoV-2 infection in the different cohorts.
- To identify potential biomarkers of increased susceptibility to virus/smoke-mediated toxicity.

Significance

Determining the impact of exposure to air pollution on SARS-CoV-2 infection in different age and health cohorts will provide a clear scientific basis for targeted public health management in areas of high air pollution especially during the expanding bushfire seasons. It also offers a new platform to identify biomarkers to elucidate the impact of combined SARS-CoV-2 and air pollution on high-risk populations.

Results

- Olfactory cells contain SARS-CoV-2 entry receptors.
- Olfactory cells from younger individuals are more resilient to co-treatment with Air pollutants and SARS-CoV-2.

(A) Presence of ACE2 (green) and TMPRESS 2 (magenta) in positive control Vero cells.

(B) Presence of ACE2 and TMPPRSS2 on an olfactory cell line from our biobank.

Olfactory cells from a 17-year-old (Y) show increased viability in comparison to a 74-year-old (O) when co-treated with air pollution and SARS-CoV-2.