Why Are Air Pollutants Toxic?

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Background

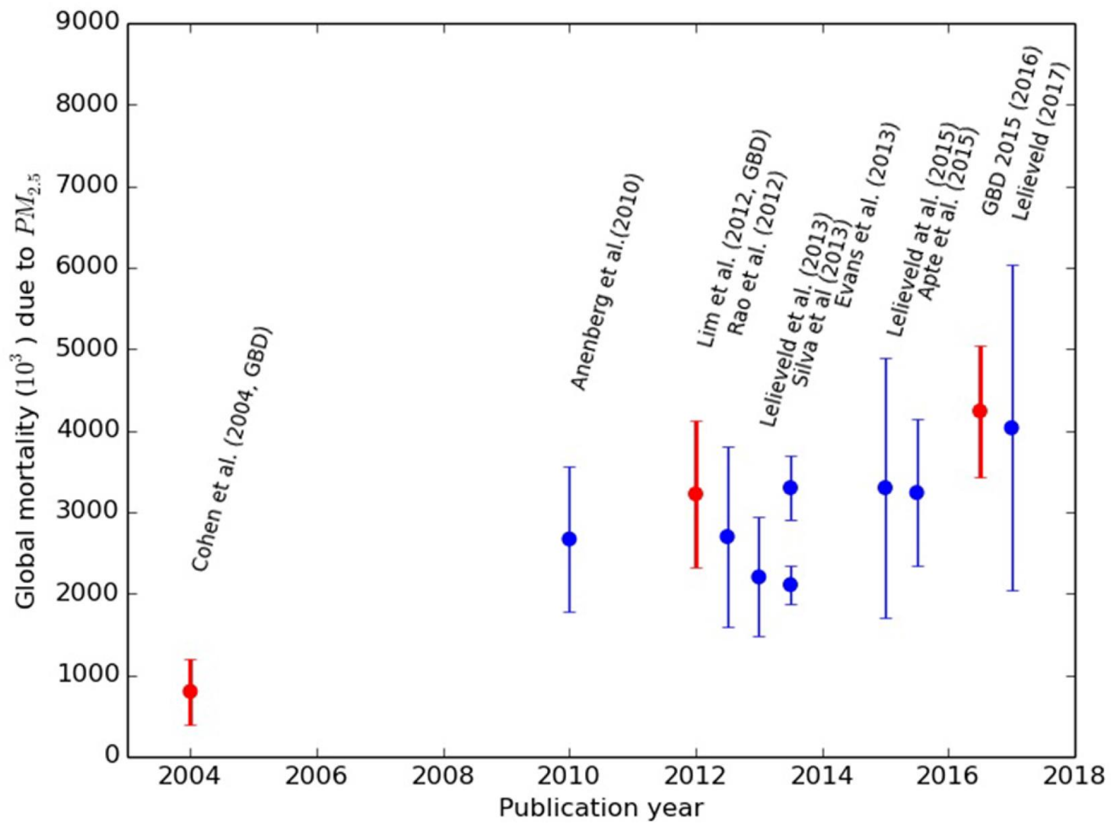

Fig. 1 Literature estimates of global premature mortality attributed to outdoor air pollution by fine particulate matter ($PM_{2.5}$). Red color indicates Global Burden of Disease review articles.

~4 million people die every year due to $PM_{2.5}$

Why is $PM_{2.5}$ toxic ?
Because $PM_{2.5}$ is so small, reaches ELF, and produces ROS therein.

What happens if OH-radicals are formed in ELF ?
Surfactant proteins will be oxidized. However, we don't know how.

- ELF = Epithelial Lining Fluid
- ROS = Reactive Oxygen Species (e.g., OH-radicals)

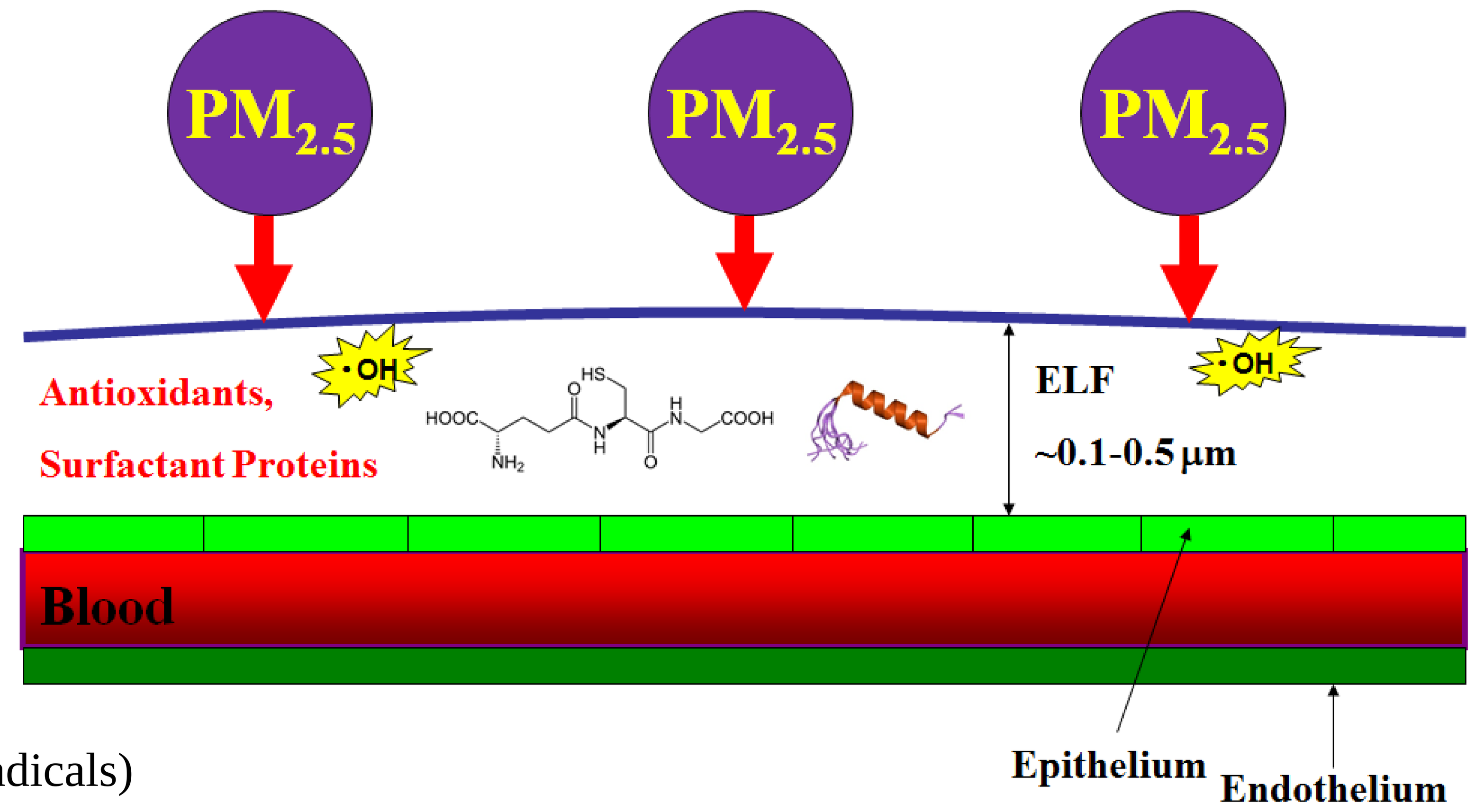

Fig. 2 Inhalation of $PM_{2.5}$ induces OH-radicals in epithelial lining fluid (ELF). ELF contains antioxidants, lipids and surfactant proteins.

References:
- S. Enami et al., "Acidity enhances the formation of a persistent ozonide at aqueous ascorbate/ozone gas interfaces" *Proc. Natl. Acad. Sci. U.S.A.*, **2008,** *105*, 7365.
- M. Shiraiwa, S. Enami et al., "Aerosol health effects from molecular to global scales", *Environ. Sci. Technol.*, **2017**, *51,* 13545.

Simulation of OH-radical oxidations of human surfactant protein B at the air-ELF interface

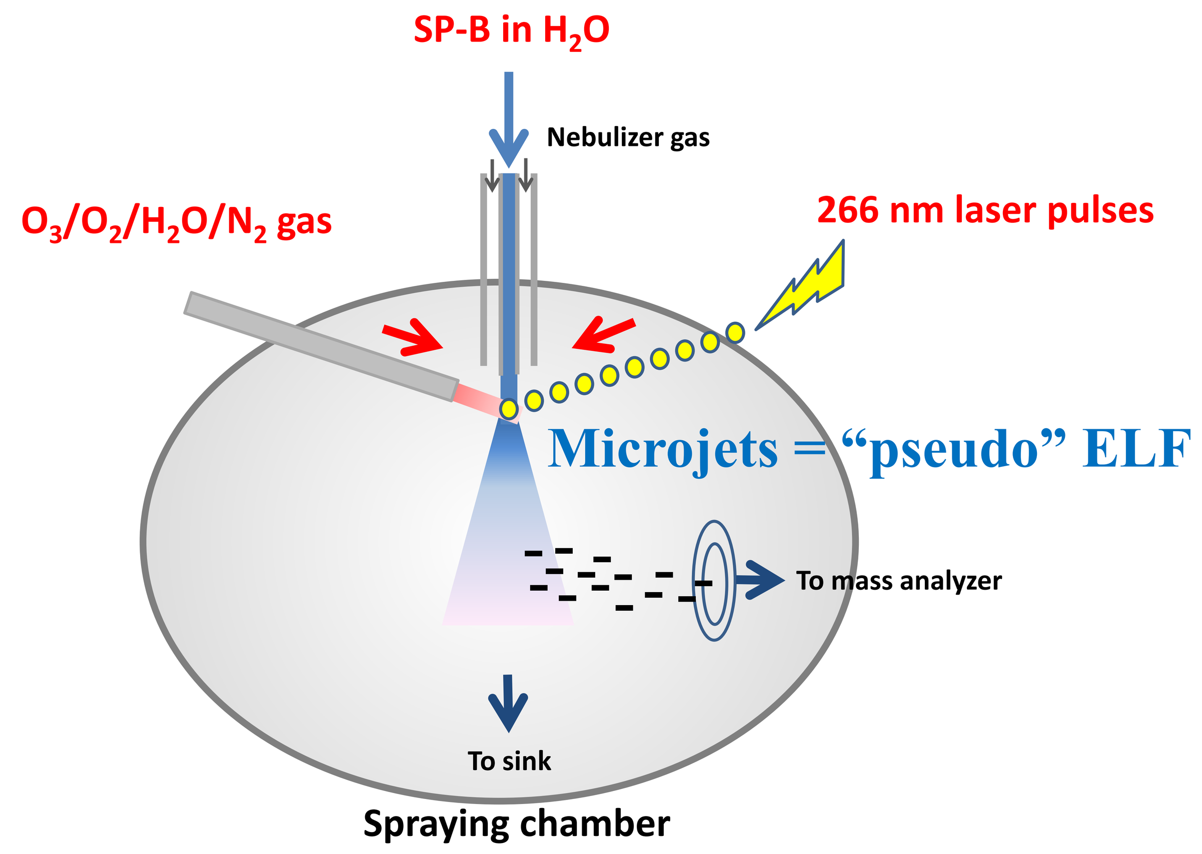

Fig. 3 Schematic diagram of present method. Water microjets containing SP-$B_{1\text{-}25}$ are exposed to gaseous OH-radicals, generated by photolysis of O_3/H_2O gas mixture.

$$O_3 + h\nu \rightarrow O(^1D) + O_2 \quad \text{(R1)}$$
$$O(^1D) + H_2O\ (g) \rightarrow 2\ OH \quad \text{(R2)}$$
$$OH(g) + \text{SP-B}_{1\text{-}25}(aq) \rightarrow \text{products } (\textbf{detected}) \quad \text{(R3)}$$

Advantages:
- **In-situ** photochemical generation of OH-radicals
- Direct detection of **interfacial** (~1 nm) intermediates within <10 µs
- Under **ambient** pressure and temperature

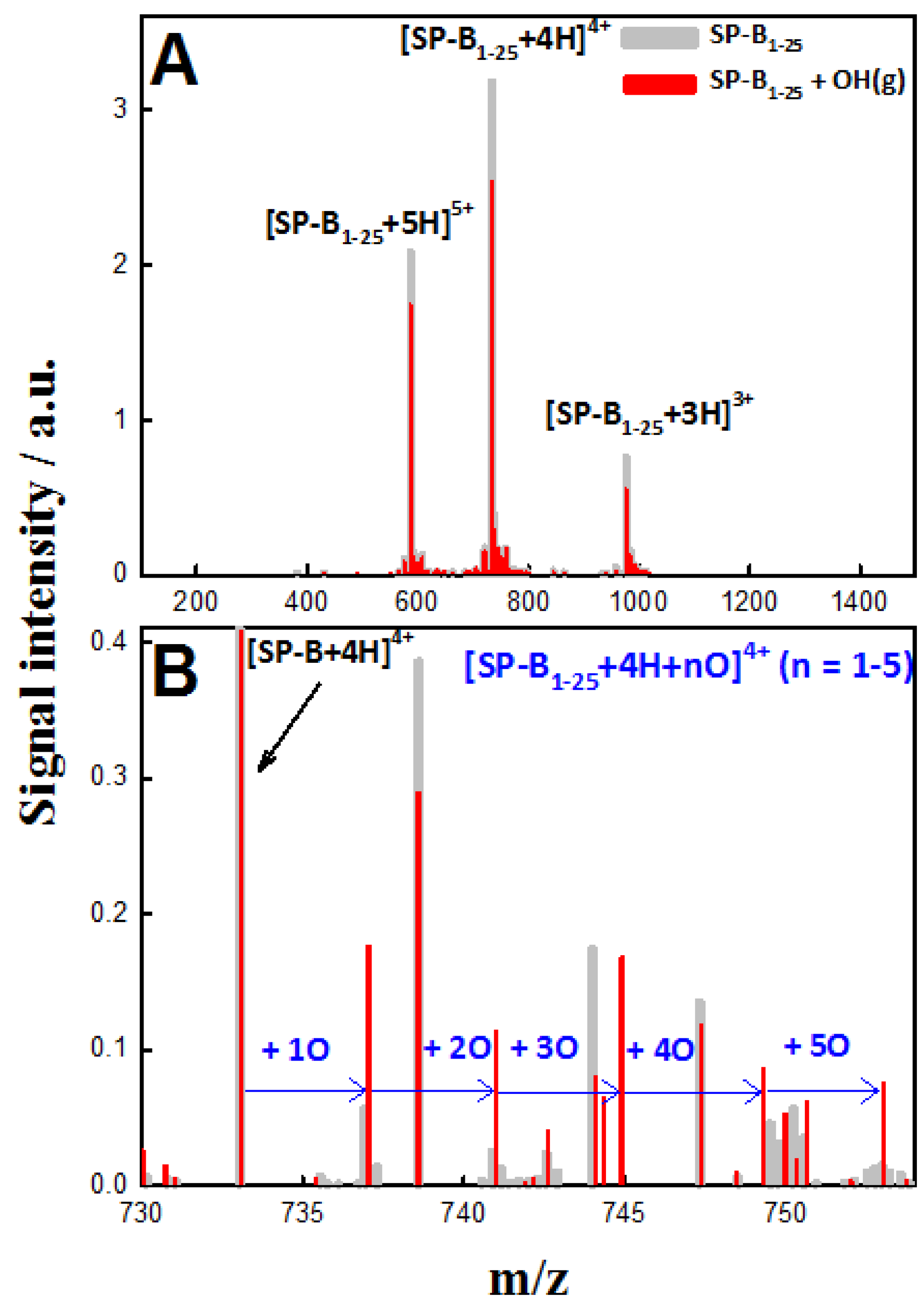

Fig. 4 A) Positive ion mass spectra of aqueous 43 µM SP-$B_{1\text{–}25}$ microjets in $O_2(g)/H_2O(g)/N_2(g)$ mixtures (gray), or exposed to ~500 ppmv $O_3(g)$ in $O_2(g)/H_2O(g)/N_2(g)$ mixtures in the presence (red) of 40 mJ 266 nm pulses. B) Spectra of $[SP-B_{1\text{–}25}+4H^+]^{4+}$ and its oxidation products in the 730–760 Da range.

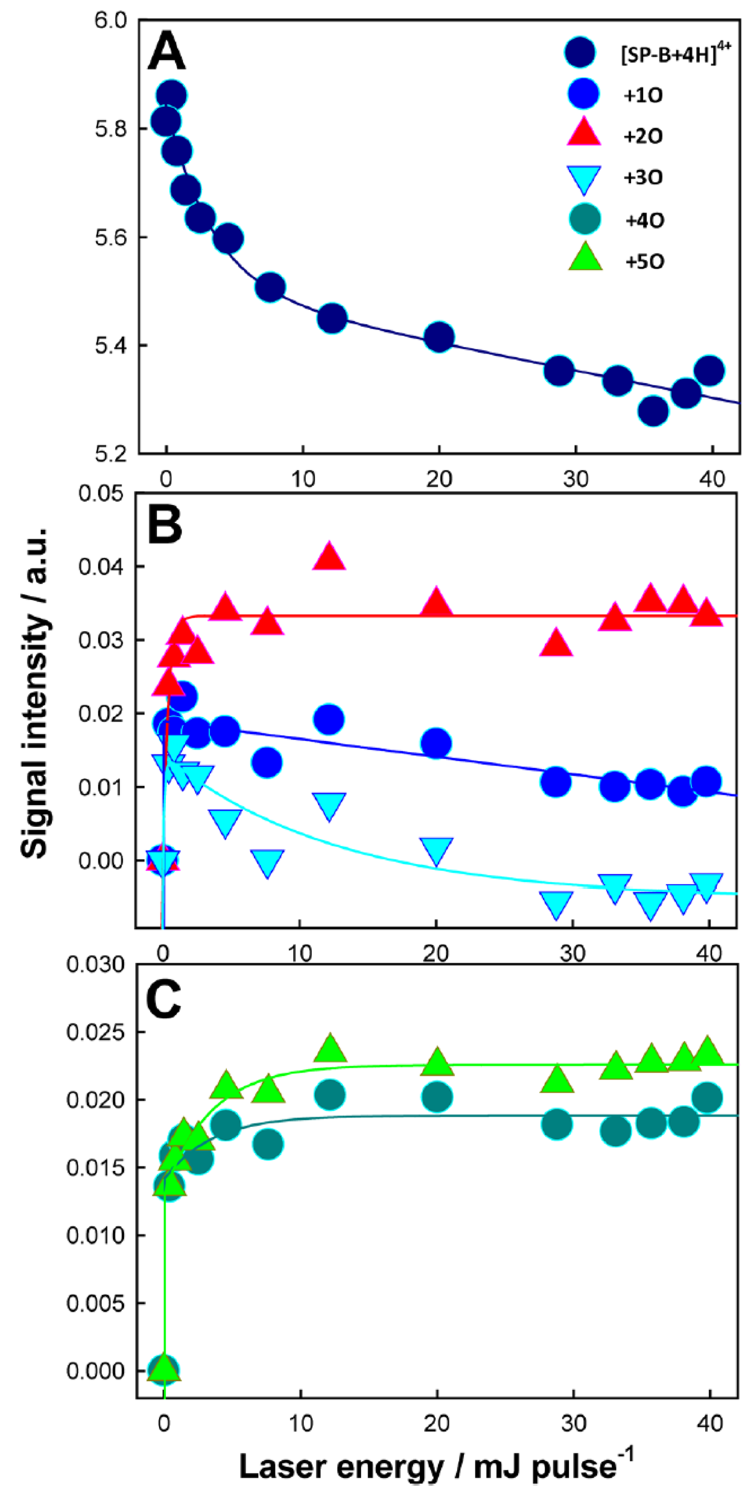

Fig. 5 Reactant (A) and products (B, C) mass spectral signal intensities from aqueous 43 µM SP-$B_{1\text{–}25}$ microjets exposed to $O_3(g)/O_2(g)/H_2O(g)/N_2(g)$ mixtures at $[O_3(g)]$ ~600 ppmv, irradiated with 266 nm laser beams as functions of laser energy (in mJ pulse^{-1}). Background (before pulsing at 266 nm) products signals were subtracted.

SP-$B_{1\text{-}25}$: $F_1P_2I_3P_4L_5P_6Y_7C_8W_9L_{10}C_{11}R_{12}A_{13}L_{14}I_{15}K_{16}R_{17}I_{18}Q_{19}A_{20}M_{21}I_{22}P_{23}K_{24}G_{25}$

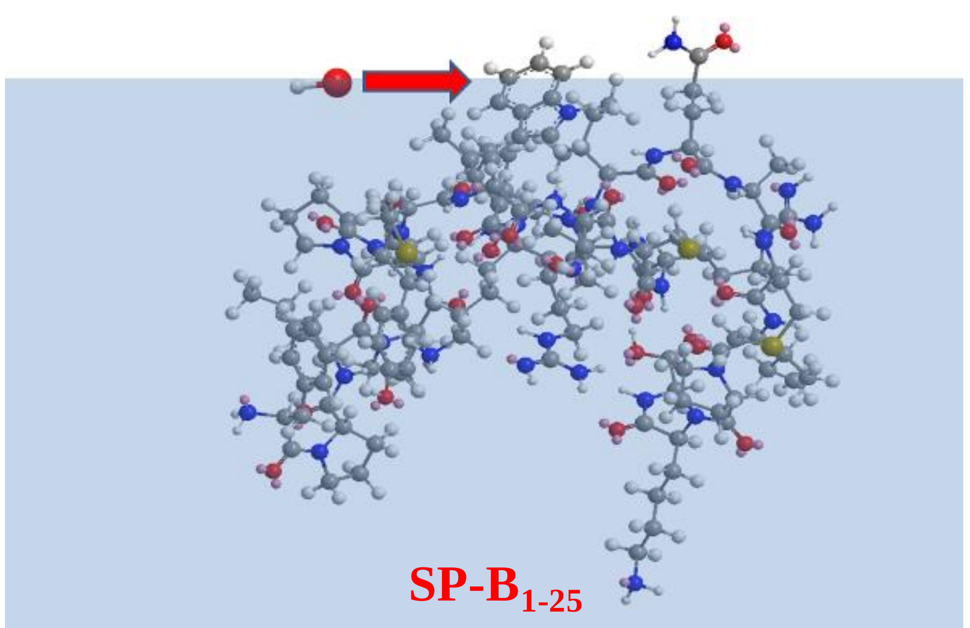

Fig. 6 OH-radicals selectively oxidize two cysteines and one tryptophan of SP-$B_{1\text{-}25}$ at the air-water interface.

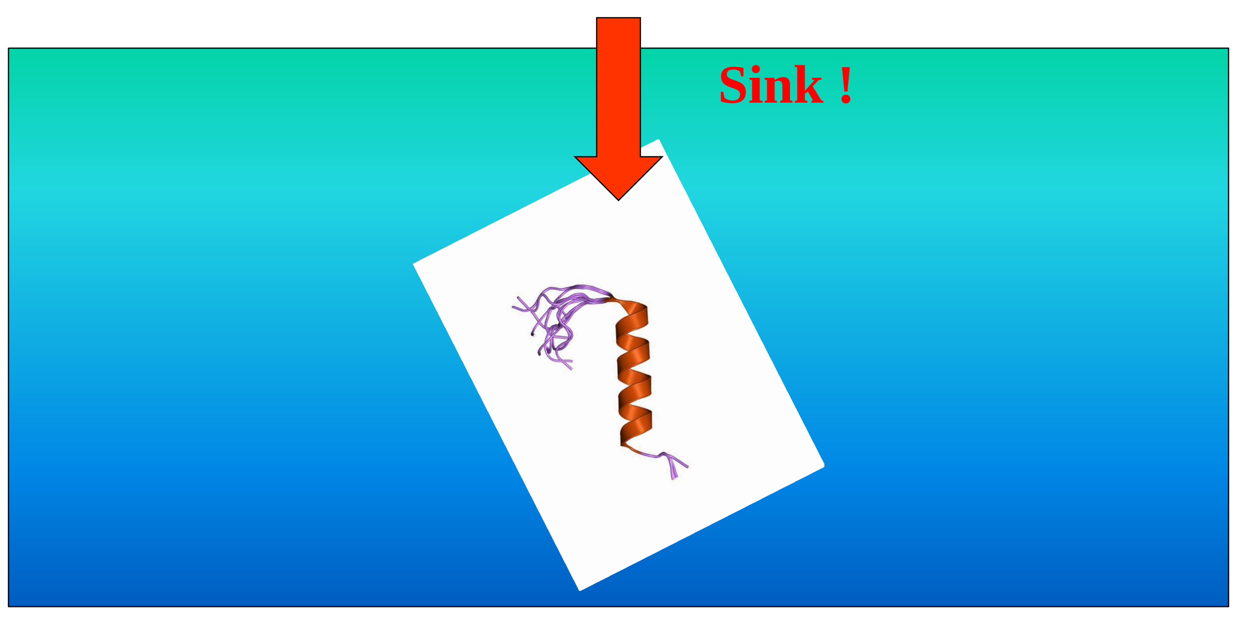

Fig. 7 $PM_{2.5}$-induced OH-radicals degrade the tensioactive properties of SP-B. This mechanism may partly explain why $PM_{2.5}$ is "toxic".

Reference: S. Enami and A. J. Colussi, "OH-radical oxidation of lung surfactant protein B on aqueous surfaces", Mass Spectrom. (Tokyo), 2018, 7, S0077.