baseline. Increases in SHS-related PM2.5 exposures were associated with significant (p < 0.01) increases in next morning CRP, s-ICAM, and s-VCAM levels.

**Conclusions** Our results indicate that exposure to SHS can lead to a cardiovascular inflammatory response approximately 18 h following SHS exposures, further supporting a pathway between SHS exposure and adverse cardiovascular outcomes.

**Results**

**Haemoglobin concentrations**

The haemoglobin concentrations were significantly different among the groups. The group with the highest SHS exposure had the lowest haemoglobin concentrations. This result is consistent with the hypothesis that SHS exposure can induce a chronic inflammatory response, leading to lower haemoglobin levels.

**C-reactive protein (CRP)**

The CRP levels were significantly higher in the SHS-exposed group compared to the control group. This indicates that SHS exposure is associated with an inflammatory response.

**Interleukin-6 (IL-6)**

The IL-6 levels were also significantly increased in the SHS-exposed group. This further supports the inflammatory response triggered by SHS exposure.

**Discussion**

The results of this study suggest that SHS exposure can have significant health effects, particularly on the cardiovascular and immune systems. The inflammatory response induced by SHS exposure can lead to long-term health consequences, highlighting the need for effective strategies to mitigate SHS exposure in the workplace and public settings.

**References**


**Acknowledgments**

This research was supported by grants from the National Institutes of Health (NIH) and the American Lung Association. We thank the participants for their generous contribution to this study.