Toxicological responses of normal human bronchial epithelium (NHBE) model exposed to settled dust samples from moisture damaged and reference schools

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AIMS

Exposure to indoor air in moisture damaged buildings is associated with deteriorating respiratory health, assumedly due to emissions from microbial growth and wet building materials. Previous studies of toxicological effects of mouldy house microbes have indicated that inflammation and cell death are important mechanisms.

METHODS

Aiming to gain further insight into function of respiratory epithelia, we studied the responses of normal human bronchial epithelium (NHBE) model after exposure to settled dust samples collected during 8 weeks from moisture damaged (n=6) and reference (n=3) schools in Spain, The Netherlands and Finland. The collected dust was vacuumed onto MCE filter, suspended in dilution buffer and stored frozen before the experiments.

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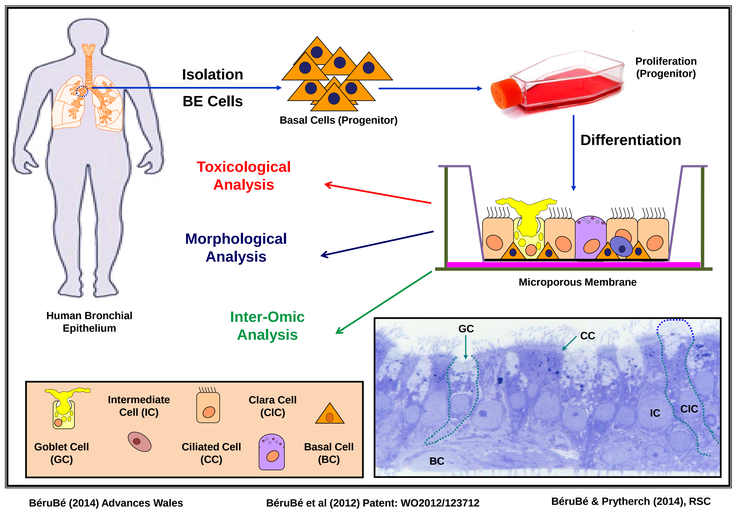

The effects of 24 hours of exposure on the NHBE model was assessed by measuring changes in mucus production (Bradford assay) and transepithelial electric resistance (TEER) of the lung epithelia. Also cell viability (ATP assay), cytokine levels in the culture media (ELISA) as well as morphological changes in cultured cells (light and scanning electron microscopy) were followed.

The results were compared with immunotoxicological potential of the same samples in mouse RAW264.7 macrophage model, where viability (MTT-assay) and production of inflammatory mediators (NO, IL-6, TNF, MIP2) were analysed after 24 hour exposure to settled dust.

ACKNOWLEDGEMENTS

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RESULTS

The results showed that exposure to dust from moisture damaged schools was capable of increasing mucus production and decreasing viability and transepithelial resistance indicating deterioration of tissue integrity and cell death in human lung tissue construct. Similarly to the results from mouse macrophage model, Dutch and Spanish samples were generally more toxic compared to samples from Finland. However, the results are only indicative due to low number of samples and high variation between the studied schools.

CONCLUSIONS

The findings suggest that the defence mechanisms present in respiratory epithelia are activated by dust from moisture damaged buildings, and in some cases the exposure may damage the affected lung tissue. The bioreactivity of the settled dust is dependent also on the location of the building.

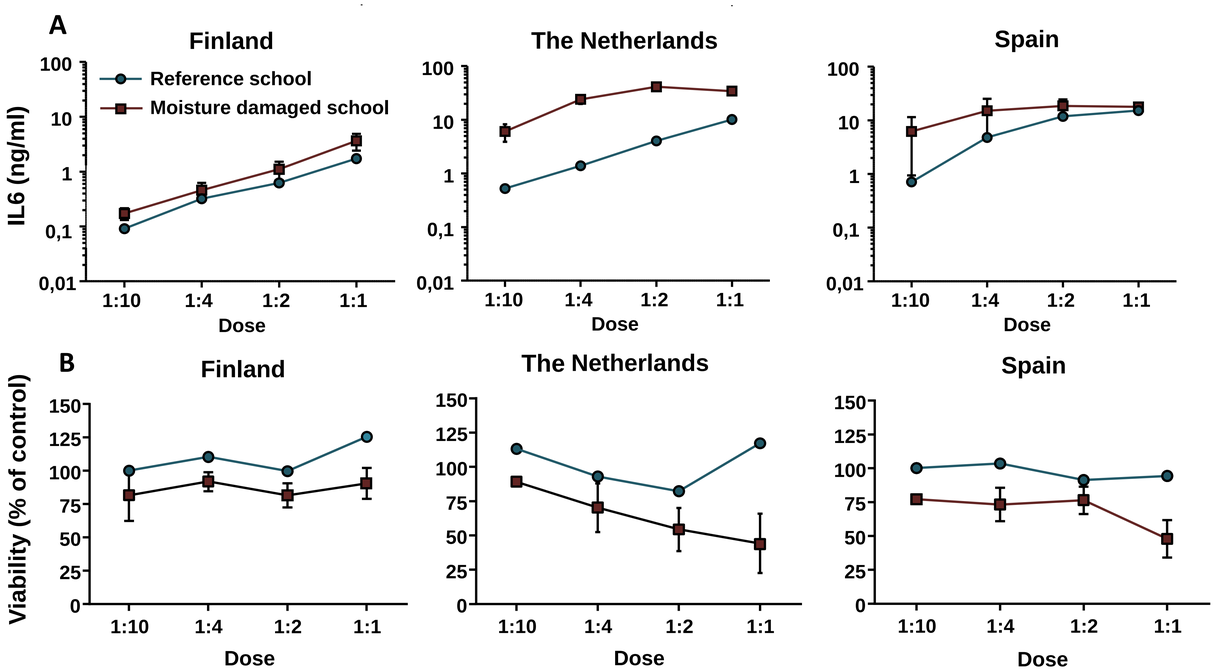

Figure 1. A) *Comparison of dose-response curves of interleukin (IL) -6 production in mouse RAW264.7 macrophages and* ***B)*** *viability of NHBE cells after exposure to settled dust from moisture damaged and reference schools in three European countries.*