IMPACT OF AIR POLLUTION ON SKIN AND RELATED PROTECTION BY A THERMAL SPRING WATER

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INTRODUCTION

In epidemiological studies, it has been shown that exposure to airbone, traffic-related particulate matter (PM) is associated with increased skin sensitivity and increased signs of skin aging including pigment spot formation. It has been observed that particles such as fine carbon black (Huber 990) and diesel exhaust particles such as SRM1650 and SRM2975 have the capacity to induce pro-inflammatory reactions in human keratinocytes leading to the production of soluble inflammatory mediators (IL-1, IL-6) and skin pigmentation markers (POMC). Previous studies also indicate that UV radiation and pollution might result in synergetic effects. La Roche-Posay thermal spring water (LRP TSW) has been successfully used for years in patients suffering from chronic inflammatory diseases. The aim of this study was to evaluate whether LRP TSW was able to inhibit or decrease pollution and UV+pollution induced damage in human keratinocytes.

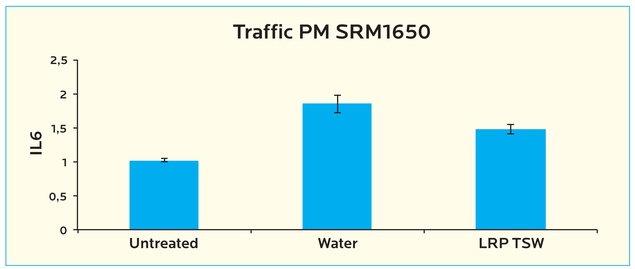
MATERIALS AND METHODS

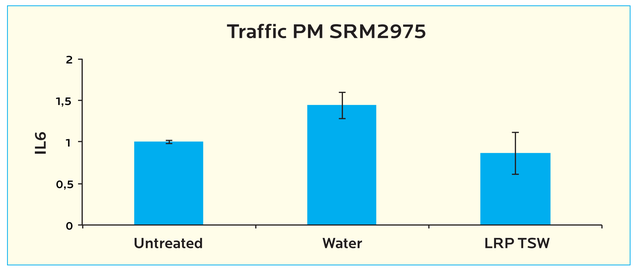
Gene expression of keratinocytes cultured in cell culture medium MCDB153 (Biochrome) dissolved in LRP TSW was compared to the results of keratinocytes cultured in the same medium prepared with deionized drinking water. 24h to 48h after exposure to pollution particles or particles + UV, total RNA isolation was performed and PCR reactions were carried out. As readout, mRNA expression of IL1α/18S rRNA, IL6/18S rRNA and POMC/18S rRNA were measured. Pollution particulate matters (PM) were diesel exhaust particles (DEP) SRM1650 and SRM2975 or fine carbon black (Huber 990). Exposure to UV radiation was performed either under a total UV spectrum (UVB+UVA/solar simulated radiation SSR) or under a UVA1 (long UVA 340-400nm) spectrum.

RESULTS

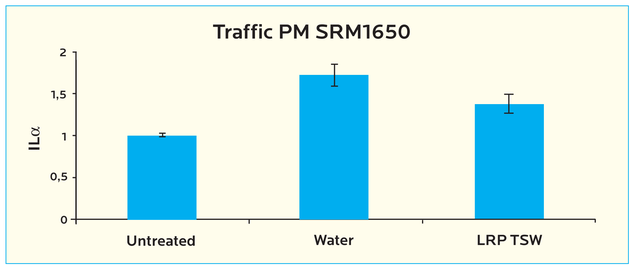
LRP TSW protection from pollution particles

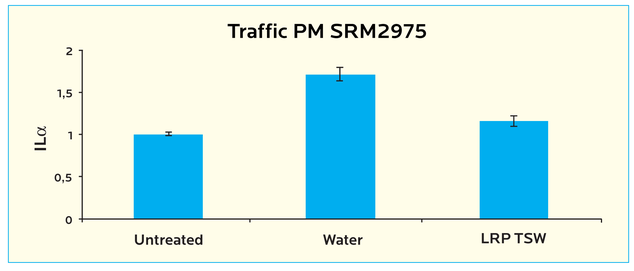
At 24h, we observed a significant ($p<0.05$) up-regulation of the pro-inflammatory marker IL6 expression when keratinocytes were exposed to SRM1650 and SRM2975 particles. LRP TSW significantly ($p<0.05$) protected from this up-regulation by respectively 43% and 100%.

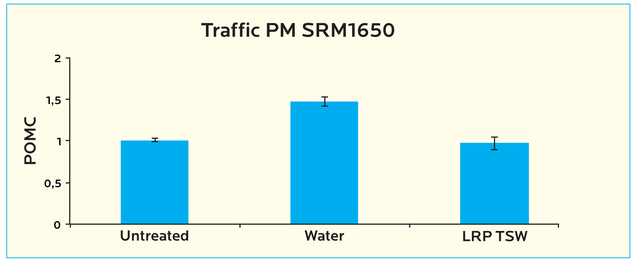




At 48h, we observed a significant up-regulation of the pro-inflammatory marker IL1α expression when keratinocytes were exposed to SRM1650 and SRM2975 particles. LRP TSW significantly ($p<0.05$) protected from this up-regulation by respectively 42% and 53%. We also observed a significant up-regulation of the pigmentation marker POMC with SRM1650 particles and that LRP TSW was able to significantly ($p<0.001$) inhibit this up-regulation.

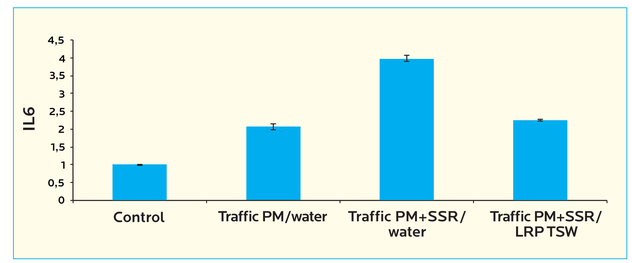




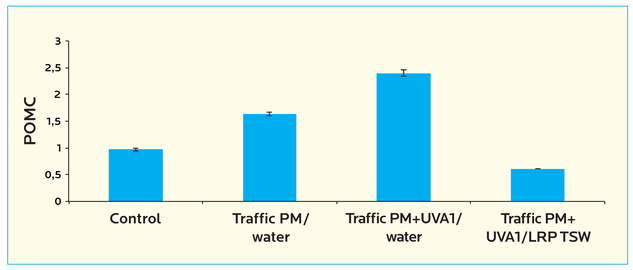


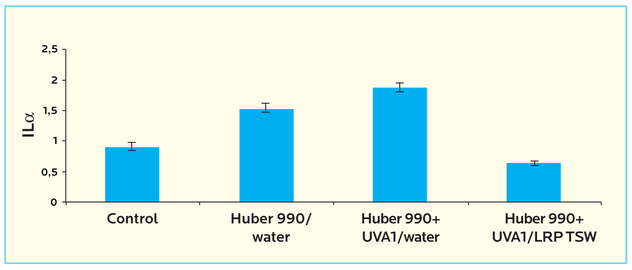
LRP TSW protection from pollution particles + UV exposure

Keratinocytes were exposed to traffic PM SRM2975 alone and to the combination of SRM2975 and SSR. There was an increased response in IL6 gene expression due to the combination of traffic PM and SSR exposure compared to traffic PM alone. LRP TSW significantly ($p<0.001$) prevented this synergetic effect.



When keratinocytes were exposed to the combination of PM SRM2975 and UVA1 (long UVA) (10J/cm²) a significant up-regulation of POMC was observed as well as a significant protective effect ($p<0.001$) by LRP TSW. With the combination of PM Huber 990 and UVA1 (long UVA) exposure (10J/cm²) a significant up-regulation of IL1α was observed as well as a significant protective effect ($p<0.001$) by LRP TSW.





CONCLUSION

This study confirms that pollution particles are able to induce inflammatory and pigmentation mediators in human keratinocytes and that the combination of these pollution particles with UV radiation has a synergetic effect on the mediators induction. We demontrasted that LRP TSW was able to protect against the inflammatory and pigmentation mediators up-regulation by traffic particulate matter alone or in combination with UV radiation. These data confirm the interest of the LRP TSW in the prevention of skin reactivity increase by pollution.

REFERENCES

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