

PIGMENTATION

LINKING AIR POLLUTION-INDUCED IMMUNE ACTIVATION TO PIGMENTATION DISORDERS

T.k. $Chan^{(1)} - L.$ $Denat^{(2)} - N.$ $Bourokba^{(1)} - P.$ $Perez^{(2)} - Jp.$ $Belaidi^{(2)} - J.$ $Li^{(3)} - P.$ $Bastien^{(2)} - C.$ $Chollet^{(2)} - D.$ $Bramono^{(1)} - J.$ $Soeur^{(2)} - S.$ $Nouveau^{(2)} - L.$ $Marrot^{(2)} - S.$ $Lim^{(1)}$

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Introduction: Air pollution has been associated with skin aging, inflammatory and allergic skin conditions.

Objective: To investigate the underlying mechanism of pollution-induced skin pigmentation disorders.

Materials and Methods: A clinical study was conducted to investigate the impact of chronic pollution on skin of a young population of women from two Chinese cities with different levels of pollution. To study the effect of pollutants on skin, primary human melanocytes were directly exposed to polycyclic aromatic hydrocarbons (PAHs) at nanomolar concentration range, in the presence or absence of UVA1. To investigate the biological impact of pollutants on systemic immunity, peripheral blood mononuclear cells (PBMCs) were exposed to PAHs and cytokine multiplex analysis was performed. Melanocytes were treated with conditioned medium from PAH-exposed PBMCs to study the biological consequences of systemic immune activation on pigmentation.

Results: Urban pollution was associated with a higher prevalence of spread macules on the cheeks, particularly visible in women > 35 years old, and a higher prevalence of simplex lentigo on the cheeks mainly in women aged 25-29 years old. When melanocytes were exposed to PAHs, no significant increase in tyrosinase activity and melanin content was observed. Primary human keratinocytes treated in the same conditions did not produce more POMC or endothelin-1. These results suggested that pollution-associated hyperpigmentation disorders may involve other paracrine signals arising from microenvironment. Traces of PAHs detected in the blood circulation of inhabitants of polluted cities suggest that pollutants enter human body via the systemic route. When PBMCs were exposed to PAHs, the level of IL-8, TNF- α , IFN- and GM-CSF were upregulated, with distinct activation profiles. Most importantly, melanocytes treated with conditioned medium from PAH-exposed PBMCs, showed a dysregulated expression of multiple pigmentation-associated genes and proteins.





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Conclusions: This study proposes a novel role for systemic immunity in regulating chronic urban pollution-related pigmentation disorders.



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