日本香粧品学会誌　Vol. 37, No. 1, pp. 6–10（2013）

〈教育セミナー〉

シワのサイエンス～成因から改善アプローチまで～

環境因子とシワ

森 田 明 理

Skin Aging and Environmental Factors

Akimichi MORITA

Abstract

Skin aging following repeated exposures to ultraviolet（UV）irradiation and tobacco smoking results largely from the damage to cutaneous connective tissue, which is composed of collagen, elastin, and proteoglycans. The alterations of cellular component and the extracellular matrix of dermal connective tissue after exposure to UV and tobacco smoke extracts. Our findings indicated that the skin premature aging induced by UV and tobacco smoke extracts share molecular features including abnormal regulation of extracellular matrix deposition through elevated MMPs, reduced collagen production and abnormal proteoglycan accumulation *via* ROS generation. Tobacco smoke contains more than 3800 constituents, including numerous water insoluble polycyclic aromatic hydrocarbons that trigger the aryl hydrocarbon receptor（AhR; also called the dioxin receptor）signaling pathway. AhR knockdown abolished the increase in transcription of the AhR-dependent gene CYP1A1/CYP1B1 and MMP-1 upon treatment with either tobacco smoke extract. These findings suggest that the tobacco smoke extracts induced MMP-1 expression in human fibroblasts and keratinocytes *via* activation of the AhR pathway. Thus, the AhR pathway may be pathogenetically involved in extrinsic skin aging. In addition, natural sun light includes visible light and infrared A（IR-A）. Although the effects of ultraviolet for photoaging have been largely investigated, those of IR-A remain unclear. Based on the recent studies, infrared A shares the mechanisms for photoaging with UV, including the induction of matrix metalloproteinase（MMP）-1 through ERK and p38 MAP kinase activation. IR-A is absorbed intramitochondrially. There might be a chromosphere for IR-A radiation.

Key words:　environmental factors, skin aging, tobacco smoking, ultraviolet, aryl hydrocarbon receptor.