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〈一般論文〉

UVA 照射で生じる培養ヒト皮膚由来線維芽細胞の石灰化機序と石灰化に対する海洋深層水の抑制作用に関する初期検討

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Preliminary Study on the Calcification Mechanism of Normal Human Fibroblasts Irradiated UVA and the Suppressive Effect of Deep Seawater on the Calcification

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Abstract

It has been reported that normal human dermal fibroblasts (NHDF) are calcified by UVA irradiation. In the arterial calcification, it is well-known that the calcification of vascular smooth muscle cells (VSMC) is accompanied by the apoptosis induced by endoplasmic reticulum (ER) stress. From these findings, we considered that calcification by UVA irradiation is more likely to be caused by a similar mechanism to that of arterial calcification. This study aimed to investigate the relationship between the apoptosis induced by ER stress and the calcification caused by UVA irradiation. Molecular biological and cell physiological methods were used to achieve the aim in this study. As a result, the calcification and the activity of caspase-3, a mediator of apoptosis were suppressed by the ER stress inhibitor 4-phenylbutyric acid (4-PBA), in UVA-irradiated NHDF. Furthermore, in UVA-irradiated NHDF, upregulation of the expression of ER stress-induced apoptosis mediators were confirmed. It has also been reported that deep seawater (DSW) suppresses this calcification. Hence, the suppressive mechanism of DSW was investigated through the relationship between the apoptosis induced by ER stress and the calcification caused by UVA irradiation. As a result, DSW suppressed the caspase-3 activity and calcification, similar to 4-PBA. In addition, DSW reduced the expression of genes specific for ER stress-induced apoptotic pathway. To summarize these results, it was suggested as a possibility that the calcification of NHDF caused by UVA irradiation is accompanied by the apoptosis induced by ER stress, and DSW regulates ER stress-induced apoptosis.

Key words:　endoplasmic reticulum stress, calcification, fibroblasts, UVA, DSW.