〈シンポジウム〉
(紫外線と皮膚を考える)

紫　外　線　発　癌

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Ultraviolet Carcinogenesis

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Abstract

The purpose of this study is to ask what kinds of DNA damage is involved in UV carcinogenesis. Firstly, we produced UV induced skin cancers on hairless mice and *ras* gene alterations in UV induced mice skin cancers were analyzed whether there is UV specific gene alteration. Five types of base changes including activated *ras* were detected in 9 UV-induced skin tumors. No specific prevalent gene or prevalent mutation sites were shown. However, unexpectedly, transversions were predominant, whereas previous findings indicates UVC induced mutations in shuttle vectors are predominated by transition type mutation. Although pyrimidine dimers may be involved in UV carcinogenesis, photoproducts other than dimers and (6-4)photoproduct such as 8-OhdG should be taken into consideration to see the effect by UVB *in vivo*, with the presence of endogenous photosensitizer. Secondly, we detected mutations in *p 53* and *Ras* genes of skin cancers from patients with xeroderma pigmentosum (XP), having deficiency in the excision repair. Fifty persent of Non-Melanoma-Skin Cancers (NMSC) from XP patients had mutations at *p 53* gene. The mutation occurred preferentially at CC site and transitions were predominant in *p 53*, whereas *ras* gene mutations was far less frequent over the same samples, which implies that DNA damage caused by sunlight rarely hit the crucial sites of *ras* gene. Lastly, *p 53* gene mutations of NMSC were compared between sun-exposed area and un/less exposed area. The frequency of the *p 53* gene mutations between the two groups were almost the same. However, in skin cancers from sun-exposed area, 67% had the transition at dipyrimidine sites, whereas only 20% had the same type of mutations from un/less exposed area and this difference was statistically significant ($p < 0.05$).

Key words: UV carcinogenesis, *p 53, ras*, transition, transversion.