

Are (6-4) photoproducts the main ultraviolet-induced lethal lesions?

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Are (6-4) photoproducts the main ultraviolet-induced lethal lesions?

Research Project

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02680167

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Grant-in-Aid for General Scientific Research (C)

Allocation Type

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代謝生物化学

Research Institution

Nara Medical University (1991)
Kanazawa University (1990)

Principal Investigator

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cyclobutane dimer / (6-4) photoproduct / monoclonal antibody / XP revertant / xeroderma pigmentosum / UV / DNA repair / ELISA

Research Abstract

Cleaver et al. have established UV-resistant xeroderma pigmentosum (XP) revertant cells from UV-sensitive XP-A cells by chemical treatment. Surprisingly, UV-resistant XP revertant cells are still deficient in the repair of cyclobutane pyrimidine dimers, although they have recovered the repair ability of (6-4) photoproducts, suggesting that (6-4) photoproducts are the main UV-induced lethal lesions. To confirm these results, I examined UV-induced cytotoxicity in XP-A, XP revertant and normal human cells by colony formation method. XP-A cells were 10 times as UV-sensitive as normal cells. I found that XP revertant cells had obtained almost normal UV sensitivity as reported by Cleaver et al. Next, the repair of two types of DNA damage (cyclobutane dimers and (6-4) photoproducts) was examined by the sensitive ELISA using monoclonal antibodies, which I had newly established, against photolesions. In the repair of (6-4) photoproducts, XP-A repaired only 30% within 24 hr after irradiation (10 J/m^2), while normal cells repaired more than 90% within 3 hr. XP revertant showed almost normal repair pattern. In the repair of cyclobutane dimers, XP-A, XP revertant and normal cells repaired 20%, 40% and 60% within 24 hr after irradiation, respectively. I confirmed Cleaver's results showing that XP-revertant had almost normal repair on (6-4) photoproducts, but had reduced repair on cyclobutane dimers. However, I found that XP revertant did have reduced repair on cyclobutane dimers, but not completely inhibited repair reported by Cleaver et al. The results were confirmed by the repair experiment using low UV dose (2 J/m^2). These results suggest that XP revertant cells still have some residual repair capacity for cyclobutane dimers. Thus, these results suggest that cyclobutane pyrimidine dimers are not excluded as a candidate for the main UV-induced lethal damage.

Research Products (14 results)

All Other

All Publications (14 results)

- [Publications] Toshio Mori: "In situ (6-4)photoproduct determination by laser cytometry and autoradiography" Mutation Res.236. 99-105 (1990) ▼
- [Publications] Tsukasa Matsunaga: "Base sequence specificity of a monoclonal antibody binding to (6-4)photoproduct" Mutation Res.235. 187-194 (1990) ▼
- [Publications] Toshio Mori: "simultaneous establishment of monoclonal antibodies specific for either cyclobutane pyrimidine dimer or (6-4)photoproduct from the same mouse immunized with ultraviolet-irradiated DNA" Photochem.Photobiol.54. 225-232 (1991) ▼
- [Publications] Tsutomu Muramatsu: "Induction and repair of UVB-induced cyclobutane pyrimidine dimers and (6-4) photoproducts in organ-cultured normal human skin" Arch.Dermatol.Res. ▼
- [Publications] Toshio Mori: "An XP complementation group A related gene:confirmation using monoclonal antibodies against the cyclobutane dimer and the (6-4)photoproduct" Mutation Res. ▼
- [Publications] C.F.Arlett: "Hypersensitivity of human lymphocytes to UV-B and solar irradiation:Evidence for a novel excisable DNA lesion" Proc.Natl.Acad.Sci.,U.S.A. ▼
- [Publications] 森 俊雄: "細胞トキシコロジー試験法 4.4DNA鎖切断の検出法" 朝倉書店, 226-237 (1991) ▼
- [Publications] 森 俊雄: "生命薬科学実験講座 V.遺伝子 DNA損傷検出法" 広川書店, ▼
- [Publications] Toshio Mori et al.: "In situ (6-4) photoproduct determination by laser cytometry and autoradiography." Mutation Res.236. 99-105 (1990) ▼
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- [Publications] C. F. Arlett et al.: "Hypersensitivity of human lymphocytes to UV-B and solar irradiation : Evidence for a novel excisable DNA lesion." Proc. Natl. Aced. Sci., U. S. A. ▼

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