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Drinking green tea prevents UVB-induced immunosuppression through rapid repair of damaged DNA and enhancement of nucleotide excision repair gene expression in mice

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As UVB-induced immunosuppression has been implicated in the development of skin cancers, we investigated whether drinking green tea polyphenols (GTPs) can modulate the effects of UVB radiation on the immune system. Treatment of C3H/HeN mice with GTPs in drinking water inhibited UVB-induced suppression of contact hypersensitivity (CHS) response which was associated with the enhancement of the levels of IL-12. Intraperitoneal injection of mice treated with GTPs with neutralizing anti-IL-12 antibody abrogated the protective effects of GTPs against UVB-induced suppression of CHS. The administration of GTPs did not prevent UVB-induced suppression of CHS response in IL-12 knockout (IL-12 KO) mice but prevent it in their wild-types. As UV-induced DNA damage, particularly in the form of cyclobutane pyrimidine dimers (CPDs), has been implicated in UV-induced immunosuppression, we determined the effect of GTPs on the repair of UV-induced CPDs in mouse skin. GTPs repaired or reduced the number of UVB-induced CPD⁺ cells in the skin of wild-type mice more rapidly compared to non-GTPs-treated wild-type mice. In contrast this effect of GTPs was not observed in IL-12 KO mice. However, the numbers of UVB-induced CPD⁺ cells were significantly reduced in IL-12 KO mice when GTPs-treated mice were s.c. injected with rIL-12 on UV-exposed site. Real-time PCR analysis revealed that the levels of mRNA expression of nucleotide excision repair (NER) genes [*XPC*, *XPA*, *DDB1* and *RPA* (p14)] in GTPs-treated UV-exposed wild-types were higher than non-GTP-treated UV-exposed wild-types. This effect of GTPs was not observed in IL-12 KO mice. The treatment of NER-proficient mice with GTPs promoted repair of UVB-induced CPDs but not in NER-deficient mice, together suggesting that NER mechanism is involved in GTPs-mediated DNA repair.