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The green tea polyphenol (−)-epigallocatechin gallate and green tea can protect human cellular DNA from ultraviolet and visible radiation-induced damage

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Background: **Antioxidant compounds in green tea may be able to protect against skin carcinogenesis and it is of interest to investigate the mechanisms involved. A study was therefore conducted to determine whether the isolated green tea polyphenol (−)-epigallocatechin gallate (EGCG) could prevent ultraviolet radiation (UVR)-induced DNA damage in cultured human cells. This work was then extended to investigate whether drinking green tea could afford any UVR protection to human peripheral blood cells collected after tea ingestion.**

Methods: **The alkaline comet assay was used to compare the DNA damage induced by UVR in cultured human cells with and without the presence of EGCG. The same assay technique was then employed to assess UVR-induced DNA damage in peripheral leucocytes isolated from 10 adult human volunteers before and after drinking 540 ml of green tea.**

Results: **Initial trials found that EGCG afforded concentration-dependent photoprotection to cultured human cells with a maximal activity at a culture concentration of 250 μM. The cells types tested (lung fibroblasts, skin fibroblasts and epidermal keratinocytes) demonstrated varying susceptibility to the UVR insult provided. The *in vivo* trials of green tea also demonstrated a photoprotective effect, with samples of peripheral blood cells taken after green tea consumption showing lower levels of DNA damage than those taken prior to ingestion when exposed to 12 min ultraviolet A (UVA) radiation.**

Conclusion: **The studies showed that green tea and/or some constituents can offer some protection against UV-induced DNA damage in human cell cultures and also in human peripheral blood samples taken post-tea ingestion.**

Key words: **antioxidants; comet assay; green tea; skin cancer; ultraviolet radiation.**

There are more than 40 000 new cases of skin cancer each year in the UK. Of these, approximately 10% are malignant melanomas (MM), with a significant risk of mortality if not treated promptly (1). While there is some evidence that malignant melanoma induction may be related to ultraviolet radiation (UVR) (280–400 nm) exposure (2, 3), there is stronger evidence of a link to non-melanoma skin cancers including basal cell and squamous cell carcinomas (1). In some cases, the development of skin cancer has been associated with solar radiation-induced mutations in the gene coding for the p53 tumour suppressor protein (4–6).

The genotoxic and cytotoxic effects of solar radiation in skin are well documented (1–4, 6–8). Bulky DNA adducts such as cyclobutane pyrimidine dimers and 6–4 photoproducts are known to be induced directly by UVR below 330 nm (7) and can result in mutations (1). Exposure to ultraviolet A (UVA) radiation (320–400 nm) can induce mutable DNA base lesions (8) via indirect reactions involving reactive oxygen species (ROS) (7).

Although UVA radiation is generally considered to be less harmful to human skin than ultraviolet B (UVB, 280–320 nm), it accounts for more than 90% of the UVR reaching the earth's surface (1) and the longer wavelengths penetrate more deeply than UVB, leading to DNA photodamage in a wider range of cell types (1). Exposure to UVA radiation has been shown to cause skin cancer in laboratory animals (9) and it has also been implicated in the aetiology of melanogenesis (5).

Mammalian skin possesses a range of mechanisms that modulate the damaging effects of UV and visible (VIS) radiation, including enzymatic and non-enzymatic antioxidants to scavenge or quench ROS. High levels of UV/VIS exposure can lead to the depletion of cellular antioxidants, resulting in ROS-induced DNA