THE ROLE OF IRON CHROMOPHORES IN THE NEAR UV TO BLUE LIGHT-INDUCED OXIDATIVE STRESS IN ORDINARY CELLS

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Any environmental-either physical, chemical, or biological-factor that causes a disturbance in cellular prooxidant/antioxidant balance in favor of the former can induce oxidative modification of certain cellular components, leading to physiological dysfunction of organisms, which is defined as oxidative stress. Because the prooxidants involved in the stress are mostly reactive oxygen species such as oxy-radicals, hydrogen peroxide and singlet oxygen, that are presumably produced to a large extent in cells under high light conditions, bright sunlight could be regarded as an important oxidative stressor for organisms even including plants.

Among spectral components of sunlight incident on the earth's surface the UVB region(280-320nm), overlapping with the tail-end of DNA absorption, is generally believed to be largely responsible for the detrimental effects of sunlight.

However, the damaging effects resulting from the UV-DNA interaction are not necessarily oxidative stress by definition: further, the penetration efficiency of shorter wavelength radiation into tissues is significantly lower than that of longer wavelengths. In such context, the 'real' oxidative stress in many organisms in sunlight might be ascribed to the oxidative action of rather long wavelengths covering UVA(320-400nm) and blue light(400-500nm) regions, where most of cellular pigments have absorption bands with large extinction coefficients.

Mitochondria are highly pigmented organelles that practically render ordinary animal cells the typical colors. In plant cells, chloroplasts are another organelle in which a large number of the UVA to blue-absorbing molecules, in addition to the photosynthetic pigments, are also present. Therefore, it would be natural to assume that the primary events of photo-oxidative stress occur predominantly in these organelles. This is why we have focused on the UVA to blue light effects particularly in mitochondria and chloroplasts.

In this work, some results that support the following conclusions are to be presented. (1) Singlet oxygen is the major active oxygen species directly involved in the damaging effects of high light in mitochondria as well as in chloroplasts. (2) Nonheme iron moieties, notably iron-sulfur centers, are the most responsible for the generation of singlet oxygen, acting as endogenous photosensitizers in cells. (3) In plant cells, complex Ⅲ of the respiratory chain is the primary target of photooxidative damage to mitochondria by singlet oxygen generated from mitichondrial iron-sulfur centers. (4) The D1 protein degradation during photoinhibition is largely mediated by singlet oxygen diffused from the outside of PS Ⅱ, most likely from thylakoid iron-sulfurs.