

ELUCIDATION OF THE NATURE OF GENETIC DAMAGE FORMED IN THE PRESENCE OF THE SUNSCREENING AGENT, PARA-AMINO BENZOIC ACID, DURING IRRADIATION WITH NEAR ULTRAVIOLET LIGHT

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We have previously presented data (Hodges, Moss and Davies, 1976) obtained using a model bacterial system which indicates that the suncreening agent para-amino benzoic acid (PABA) is capable of sensitising bacterial DNA to the lethal effects of 313nm ultraviolet light. It was further shown that there was a concentration dependent balance between sensitisation and protection due to absorption by PABA of the incident radiation.

In continuing this work the effect of pH during irradiation on the degree of sensitisation was investigated. The results at lower pH (as shown in the table) indicate that PABA in its zwitter ion form can also bring about sensitisation but due to increased absorbance overall protection is produced at a lower concentration.

Inactivation of *E. coli* K12 AB2480 by 313nm UV + (0.01%) PABA at various pH's

pH	k (JM <sup>-2</sup> ) <sup>-1</sup>	Absorbance	Sensitisation Ratio
3.97	7.20 x 10 <sup>-3</sup>	1.183	2.95
4.69	9.80 x 10 <sup>-3</sup>	0.784	2.95
5.01	1.11 x 10 <sup>-2</sup>	0.649	3.01
5.42	1.18 x 10 <sup>-2</sup>	0.423	2.65
6.88	4.77 x 10 <sup>-3</sup>	0.094	1.32

As a first stage in extrapolation of the data obtained with this repair deficient model bacterial system to sunlight induced skin cancer we have used a bacterial strain with limited ability to repair DNA damage. Escherichia coli K12 AB1886 has the ability to carry out recombination repair, which is "error-prone" and is thought to be the major pathway for the induction of mutation after radiation damage.

Using this strain we have shown that 93% of the damage induced by the PABA sensitised irradiation is subject to "error-prone" repair and that the mutation rate per survivor is of the same order as that induced by unsensitised 313nm irradiation.

Hodges, N.D.M., Moss, S.H. and Davies, D.J.G. (1976) *J. Pharm. Pharmac.*, 28, suppl., 53P.