suspension. After incubation at 37 °C overight, cell counts were made to calculate the percentage survivors as shown in the Figure.

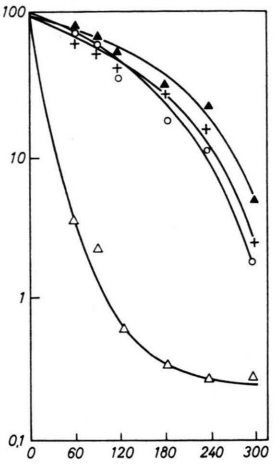

Fig. 1. Dark repair of u.v.-inactivated *E. coli* 15T⁻ in the presence of caffeine and cAMP. Survival of *E. coli* 15T in the presence of caffeine (△—△), cAMP (o—o), caffeine and cAMP (▲—▲) and the control (+—+). Ordinate: Percent survival; Abscissa: UV-dose (ergs/mm²).

As shown in the Fig. 1, caffeine strongly inhibits the excision repair part of the dark reactivation in *E. coli* 15T⁻ at the u.v. doses used. This inhibition can be reversed by cAMP. It is to be noted that in the presence of cAMP alone, the dark-reactivation directs no significant influence in the survival rates.

Recently it was reported that, in vivo [8] as well as in vitro [9], caffeine depresses the excision of pyrimidine dimers, presumably by binding to the excision enzyme involved in dark repair. The reversal with cAMP of the inhibition by caffeine in our experiments indicates that both compounds compete with the phosphodiesterase-like enzymes necessary for the removal of u.v. photoproducts, whereby cAMP binds to the enzyme with a greater affinity than caffeine.

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Photochemistry and Photobiology of 5-Ethyl- and 5-Propyldeoxyuridine

KAILASH K. GAURI, WOLFGANG RÜGER and ADOLF WACKER

Institut für Therapeutische Biochemie der Universität Frankfurt a. M.

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Thymine-, 5-ethyluracil- and 5-propyluracildeoxyriboside, irradiated with ultraviolet light (254 mμ) in a frozen aqueous solution undergo a photochemical change which can be folowed by extinction measurements at 266 mμ (Table 1). As follows from these results the absorption of thyminedeoxyriboside as well as that of 5-ethyluracildeoxyriboside increases on reirradiation (254 mμ) in water, showing that dimerization [1] has taken place. However, the magnitudes of their dimerization are different. For example, at the same ultraviolet-light dose, the dimerization of thymine-deoxyriboside goes to an extent of 30% (80—50) while that of 5-ethyluracildeoxyriboside to to 6% (77—71) only. The inability of 5-propyluracildeoxyriboside to dimerize shows that it reacts in a different manner which still remains to be studied.

Compound	Absorption at 266 mμ in per cent					
	Test solution before irradiation	After irradiation in ice	After reirradiation in water			
UV Dose [ergs/mm²·10⁻⁵]	—	2	0.2	0.4	1.2	2.4
Thyminedeoxyriboside	100	50	60	66	73	80
5-Ethyluracildeoxyriboside	100	71	76	77	76	60
5-n-Propyluracildeoxyriboside	100	73	73	72	64	49

Table 1. Relative change in absorption of thymine-, 5-ethyluracil- and 5-propyluracil-deoxyriboside after irradiation with ultraviolet light (254 mμ). Lamp: Low-pressure mercury lamp, NN 30/89 Quarzlampengesellschaft Hanau, Germany. Concentration: 10^{-4} M. Synthesis: 5-ethyluracil- and 5-propyluracil-deoxyriboside were synthesized by the method described by HOFFER et al. [8].

Reprints request to Prof. Dr. A. WACKER, Institut für Therapeutische Biochemie der Universität *D-6000 Frankfurt am Main*, Ludwig-Rehn-Str. 14.

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Experiments in this laboratory have shown that the lethal effect of ultraviolet-light on bacterial cells depend mainly, if not entirely, upon the photo-dimerization of thymine [2, 3]. Accordingly, the bacteria in which thymine has been replaced by 5-ethyluracil should show a comparative resistance against ultraviolet-light because of a lower degree of dimerization of 5-ethyluracildeoxyriboside. As illustrated in Fig. 1, *E. coli* cells grown in the presence of 5-ethyluracil have become distinctly more resistant to ultraviolet-light. As a comparison we have irradiated *E. coli* 15T⁻ cells containing 5-bromouracil, which is known to increase ultraviolet sensibility [4].

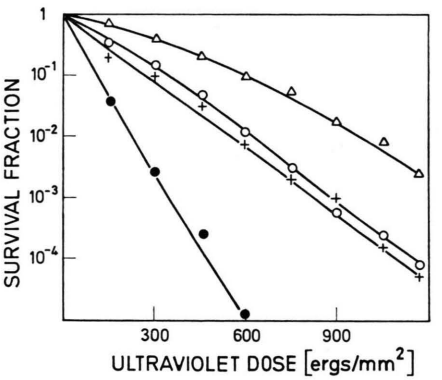

Fig. 1. Ultraviolet survival curves of *E. coli* 15T⁻ grown in the presence of 1 μg/ml thymine (+—+), 1 μg thymine + 10 μg ethyluracil/ml (△—△), 1 μg thymine + 100 μg propyluracil/ml (○—○), and 1 μg thymine + 50 μg bromouracil/ml (●—●). For composition of the culture medium see DAVIS and MINGIOLI [7].

By the use of ^{3}H-labelled ethyluracil and ^{14}C-labelled bromouracil we noticed in the experiment presented in Fig. 1 that 4.4% thymine were replaced by ethyluracil and about 40% by 5-bromouracil.

As our previous studies with a dimerization-resistant thymine analogue 5-azathymine have shown [5], the present results bring a further evidence that the in-vivo effects of these compounds can be predicted from their in-vitro dimerization by ultraviolet-light. These results confirm our previous finding that thymine is mainly responsible for the ultraviolet damage in bacterial cells.

As shown in Fig. 1, the bacteria grown in presence of 5-propyluracil are not distinctly resistant to ultraviolet-light. This may be due to the rate of incorporation of 5-propyluracil in bacterial DNA. Experiments of PIECHOWSKA and SHUGAR [6] have shown also that ethyluracil is incorporated into bacterial DNA.

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