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A role of host bacteria in inducibility of colicin I production by ultraviolet light

Colicin production can be induced in certain colicinogenic bacteria by ultraviolet irradiation[1]. OZEKI AND STOCKER[2,3] transferred colicinogenic factors E_1, E_2, and I to *Salmonella typhimurium*, strain LT2, and found that ultraviolet treatment induced an increase in production of colicins E_1 and E_2, but not of I, from bacteria carrying the corresponding colicinogenic factors.

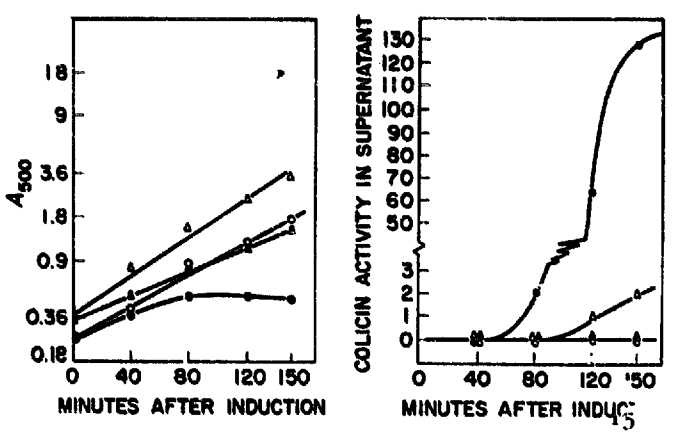
In the course of experiments on the multiplication of colicinogenic factors, we reintroduced these factors into various strains derived from *Escherichia coli* K12. *E. coli* strains carrying factor I proved to be inducible to colicin production by ultraviolet irradiation.

Release of colicin I was measured by spot-test titration on indicator strain CL104, using serial 1:2 dilutions. (The enumeration of lacunae[4] as a measurement of the number of cells induced to production of colicin cannot be used with colicin I.) The amounts of colicin I released spontaneously were roughly comparable for *S. typhimurium* LT2 and for *E. coli* K12, substrain C600. After ultraviolet treatment, there was an increase of at least 128-fold in colicin in the supernatant of *E. coli* C600 (col I), and no detectable increase with *S. typhimurium* LT2 (col I) (see Fig. 1).

The kinetics of release of colicin I from C600 (col I) after induction is similar to that reported for colicin E_1 and E_2 by FREDERICQ[5] and confirmed in the course of the present work. Another derivative of *E. coli* K12, substrain 2.0Go (col I), following similar ultraviolet treatment, releases 8 times less colicin than C600 (col I).

Evidence for vegetative multiplication of the inducible colicinogenic factors following ultraviolet irradiation has been obtained by radioisotope studies and will be reported elsewhere (P. AMATI, in preparation).

Colicinogenic factor I has the distinctive property of conferring to carrier bacteria an ability to mate with other bacteria, to transmit colicinogeny, and to act



		0.13	0.20
		1.14	1.73
		0.06	0.44
	−91	−95	−75
	2.2	4.2	6.2
	2.1	2.9	5.3
	−4	−31	−14

Fig. 1. Cells in logarithmic growth were centrifuged, resuspended in cold saline solution, and irradiated for 60 sec under a "C 50 cm distance). The suspension was diluted 1:10 in fres shaker bath at 37°. At intervals samples were taken, ths spectrophotometer at 500 mμ; after centrifugation the su membranes and assayed for colicin titer. An A_{500} va Left side: absorbancy at 500 mμ (A_{500}). Right side last active dilution). △, *S. typhimurium* (col I); ▲, S ino)-1,4-benzoquinone. ○, *E. coli* C600 (col I); ●, *E. coli* C60

as donors of chromosomal genetic markers[6,7]; in this respect factor I acts like the fertility or F factors. In *S. typhimurium*, the genetic-donor function is greatly enhanced in bacteria that have acquired factor I recently by cell contact[8]. This high-donor state is not observed in *E. coli* K12 strains carrying factor I (see ref. 7). This second difference in the behavior of factor I in *S. typhimurium* and in *E. coli* has been confirmed in the present work. The rate of contact transfer of factor I from recently infected *E. coli* K12 F⁻ bacteria is only about 10^{-5} per donor cell, compared with 0.5 for recently infected *S. typhimurium*. Ultraviolet irradiation of *S. typhimurium* (col I) and of *E. coli* K12 (col I) previous to mating did not alter the transmissibility of the colicinogenic factor.

It seems possible to explain the host-dependent differences in two functions of factor I—ultraviolet inducibility and level of contact transmission—by the following hypothesis: In *E. coli* K12 factor I is in a "stable" state, which renders it more susceptible to ultraviolet-initiated induction of vegetative multiplication, analogous to that of inducible prophages. In *S. typhimurium* factor I is more frequently "unstable" and acts more like a free fertility factor, less like an inducible prophage.

Since a state difference in *E. coli* K12 and in *S. typhimurium* is observed with colicinogenic factor I and not with colicinogenic factors E_1 and E_2, it appears that the state of a colicinogenic factor in a bacterium is controlled by the genotypes of both the factor and the host.

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