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Influence of the Fertility Episome on the Survival of X-irradiated *Escherichia coli*[1]

D. E. AXELROD[2] AND H. I. ADLER

Institute of Radiation Biology, University of Tennessee, Knoxville, Tennessee, and Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee 37830

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The resistance of *Escherichia coli* to the lethal effects of X rays was increased by the presence of a fertility episome integrated into the chromosome.

Genetic analysis has revealed several chromosomal genes and one extrachromosomal genetic factor that can influence the response of bacteria to radiation (1, 2). This report presents evidence that episome F, which controls fertility in *Escherichia coli*, can also influence resistance to the lethal effects of X rays.

From *E. coli* high-frequency Hfr donor strains (with the fertility episome integrated into the chromosome), we isolated low-frequency F^+ donors (with the fertility episome not integrated) and F^- recipients (with the fertility episome absent from the cell). Survival curves of isogenic Hfr, F^+, and F^- strains derived from strain HfrH (3) are given in Fig. 1. Similar results have been obtained with the remotely related *E. coli* lines W1485 (4) and 112-12 (6) cured of bacteriophage λ. Although the magnitude of the effect varies, within each line the Hfr strain is more resistant than its isogenic F^+ and F^- strains.

To determine whether the origin and direction of chromosome transfer affect the radiation resistance of Hfr strains, we isolated three new Hfr strains from a single F^+ strain. The origins of chromosome transfer of these Hfr strains are located on the linkage map (5) at approximately 45, 75, and 85 min, and their direction of chromosome transfer is counterclockwise, clockwise, and clockwise, respectively. The X-ray resistances of these three different Hfr strains are similar to each other but more resistant than the isogenic F^+ strain from which they were derived. Thus, the radiation resistance of Hfr strains is independent of the origin and direction of chromosome transfer.

To determine whether radiation resistance conferred by the integrated fertility episome can be transferred from Hfr donors to F^- recipients, we selected arginine-independent recombinants after a 90-min mating of strains HfrH and F^- AB1157. Since the arginine gene is located near the end of the chromosome of this Hfr donor, some of the arginine-independent recombinants would be expected to receive the fertility factor. Recombinant clones were purified, regrown, and assayed for survival after exposure to 10 kr of X rays (Fig. 2,

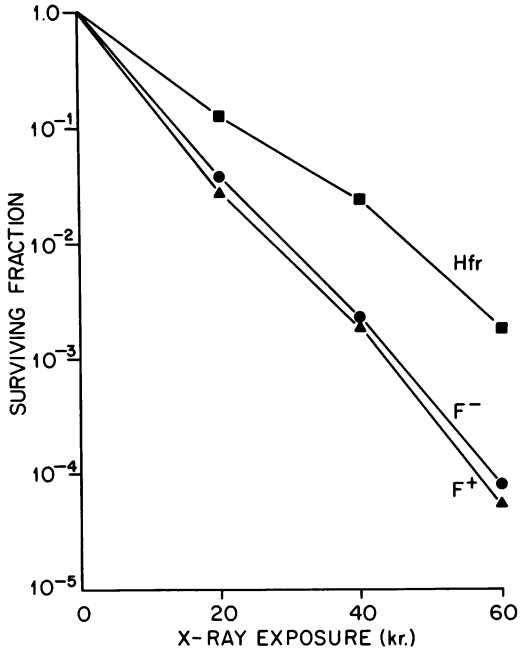

FIG. 1. *X-ray survival curves of isogenic Hfr, F^+, and F^- strains derived from HfrH. Cultures grown overnight in nutrient broth were resuspended at 5 × 10^6 bacterial/ml in phosphate buffer, exposed to 250-KV X-rays at 2.0 kr/min, and spread on nutrient agar plates. The surviving fraction is the ratio of colony-forming units/ml of irradiated sample to unirradiated sample.*

[1] These observations form part of the thesis submitted by the senior author to the University of Tennessee for the Ph.D. degree.

[2] Present address: Division of Biology, Department of Developmental Biology and Cancer, Albert Einstein College of Medicine, Bronx, N.Y. 10461.

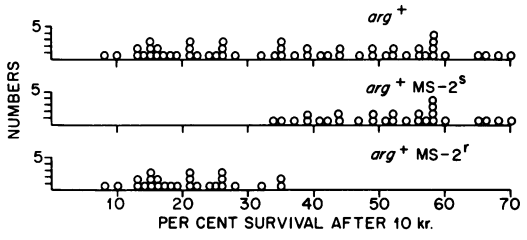

FIG. 2. *Survival, after exposure to X rays, of arginine-independent recombinant clones from a mating of HfrH and F⁻ AB1157. Each recombinant was assayed for survival after exposure to 10 kr of X rays and then was grouped according to its ability to act as a host for the donor-specific bacteriophage MS-2. Sensitivity to this bacteriophage indicates the presence of the fertility factor.*

arg⁺) and for the presence of the fertility factor by determining sensitivity to the bacteriophage MS-2. Those recombinants which have received the fertility factor (Fig. 2, *arg*⁺ MS-2ˢ) are more radiation-resistant than those which have not received it (Fig. 2, *arg*⁺ MS-2ʳ). Thus, transfer of the integrated fertility factor by conjugation is associated with an increase in radiation resistance.

We have ruled out several possible explanations for the resistance of Hfr strains: our Hfr cells do not clump, they grow at the same rate as F⁺ strains in the nutrient medium used for preirradiation growth and postirradiation plating, and they do not contain the colicin I factor or the bacteriophage λ.

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