Journal of the Arkansas Academy of Science

Volume 43 Article 36

1989

Photoreactivation of the Effect of UV Light on Gamma Ray Induced Chromosome Aberration Production in G1 Phase Xenophus Cells

Rebecca Rowe
John Brown University

H. Gaston Griggs
John Brown University

ROBISON, H.W. and N.H. DOUGLAS. 1977. Geographic distribution: *Rana sylvatica* (Wood Frog). Herpetol. Rev. 8:13.

SCHUIER, J.P., J.W. DICKSON, and M.J. HARVEY. 1972. Herpetofauna of Sylamore Range District Ozark National Forest, Arkansas: preliminary report. Arkansas Acad. Sci. 26:61-66.

SEALE, D.B. 1982. Physical factors influencing oviposition by the woodfrog, *Rana sylvatica*, in Pennsylvania. Copeia 1982:627-635.

SMITH, K.L., W.F. PELL, J.H. RETTIG, R.H. DAVIS, and H.W. ROBISON. 1984. Arkansas' natural heritage. August House Publ., Little Rock. 116 pp.

TRAUTH, S.E., M.E. CARTWRIGHT, W.E. MESHAKA, and P. CHAFFIN. 1987. Geographic distribution: *Rana sylvatica* (Wood Frog). Herpetol. Rev. 18:39.

TRAUTH, S.E., M.E. CARTWRIGHT, and W.E. MESHAKA. 1989. Winter breeding in the ringed salamander, *Ambystoma annulatum* (Caudata: Ambystomatidae), from Arkansas. Southwest. Nat. 34:145-146.

TURNIPSEED, G. 1980. Geographic distribution: *Rana sylvatica* (Wood Frog). Herpetol. Rev. 11:14.

TURNIPSEED, G. 1981. Geographic distribution: *Rana sylvatica* (Wood Frog). Herpetol. Rev. 12:13.

WALDMAN, B. and M.J. RYAN. 1983. Thermal advantages of communal egg mass deposition in wood frogs *(Rana sylvatica)*. J. Herpetol. 17:70-72.

WALTERS, B. 1975. Studies of interspecific predation within an amphibian community. J. Herpetol. 9:267-279.

STANLEY E. TRAUTH, MICHAEL E. CARTWRIGHT, and WALTER E. MESHAKA, Dept. of Biological Sciences, Arkansas State University, State University, AR 72467; Arkansas Game and Fish Commission, HC 71, Box 171, Mountain View, AR 72560; Dept. of Zoology, Oklahoma State University, Stillwater, OK 74078.

PHOTOREACTIVATION OF THE EFFECT OF UV LIGHT ON GAMMA RAY INDUCED CHROMOSOME ABERRATION PRODUCTION IN G1 PHASE *XENOPUS* CELLS

Kulp *et al.* (1985) observed that samples of G1 phase A8W4 *Xenopus* cells exposed to UV (254 nm) fluences (in the range 0-8.0 J/m²) shortly before or after being exposed to 200 rads gamma ray exhibited higher frequencies of chromosome deletions and lower frequencies of chromosome exchanges than samples of A8W4 cells exposed to 200 rads gamma ray alone. However, the chromosome-break frequencies (total number of chromosome breaks leading to aberrations/total number of cells scored) observed for cells receiving 200 rads gamma ray plus UV differed little from that for cells receiving only 200 rads gamma ray. The nature of these kinetics coupled with the observation that low fluences of UV produce few (if any) breaks in *Xenopus* G1 phase chromosomes (Griggs and Orr, 1979), and the observation that pyrimidine dimers are among the more prevalent lesions induced by UV (254 nm) in chromosomal DNA (Harm, 1980) suggested the following interpretation: The UV fluences administered had relatively little effect on the chromosome breakage induced by the gamma ray exposures, but did induce pyrimidine dimers in or near the gamma ray-break sites that significantly inhibited rejoining and restitution of broken ends of chromosomes. We describe here our initial test of this interpretation. This test was suggested by the fact that *Xenopus* cells efficiently photoreactivate pyrimidine dimers induced in chromosomal DNA by UV (Griggs and Bender, 1972, 1973; Griggs and Payne, 1981). Specifically, it was reasoned that the interpretation would appear valid if the following two questions could be answered in the affirmative: (1) Do the aberration frequencies (exchange frequency and deletion frequency) exhibited by early G1 phase cells exposed to 200 rads gamma ray + 8.0 J/m² UV + appropriate photoreactivating (PR) light fluences lie between the frequencies exhibited by such cells exposed to 200 rads gamma ray and the frequencies exhibited by such cells exposed to 200 rads gamma ray + 8.0 J/m² UV? (2) Do the chromosome-break frequencies exhibited by early G1 phase cells exposed to 200 rads gamma ray + 8.0 J/m² UV + appropriate PR light fluences lie between the frequency exhibited by such cells exposed to 200 rads gamma ray and the frequency exhibited by such cells exposed to 200 rads gamma ray + 8.0 J/m² UV?

The results of experiments performed to answer questions 1 and 2 are displayed in Fig. 1 and Table 1. Conventional techniques for cell culturing (Griggs and Bender, 1972), cell synchronization irradiations (Griggs and Orr, 1979; Cross and Griggs, 1978), preparation of metaphase spreads and aberrational analysis (Wolff, 1961) were employed. In each experiment, the starting point was the preparation of two sets of synchronous cultures of G1 phase cells (sets 1 and 2). Both sets were then irradiated as indicated (Table 1) and set 1 cultures were used to establish a detailed post-irradiation mitotic index (MI) curve. The MI curve described the post-irradiation time range (corresponding to the mitotic peak) for colcemid treatment of the set 2 cultures to obtain appropration samples of metaphase spreads for aberrational analysis.

The data shown in table 1 indicates a definite pattern of photoreactivation. Comparison of the results of experiments 2 and 3 clearly show that fewer deletions and more exchanges were observed after a PR fluence of 20.0 (J/m²/10³) than when no PR fluence had been administered. Similarly, results from experiments 4 through 7 lend supporting evidence that increasing fluences of PR between 20-35 (J/m²/10³) enhanced the rejoining and restitution of the broken ends of chromosomes. These data do indeed answer questions (1) and (2) in the affirmative and, thus, strongly support the interpretation presented by Kulp, *et al.* (1985).

LITERATURE CITED

CROSS, S. and GASTON GRIGGS. 1978. Ultraviolet light reactivation of gamma ray-induced lethal damage in vertebrate cells. Proc. Ark. Acad. Sci. 32:37-38.

GRIGGS, G. and M. BENDER. 1972. Ultraviolet and gamma-ray induced reproductive death and photoreactivation in a *Xenopus* tissue culture cell line. Photochem. and Photobiol. 15:517-526.

GRIGGS, G. and M. BENDER. 1973. Photoreactivation of ultraviolet-induced chromosomal aberrations. Science. 179:86-88.

GRIGGS, G. and T. ORR. Chromosomal aberrations resulting from ultraviolet exposures to G1 *Xenopus* cells. Photochem. and Photobiol. 30:363-368.

GRIGGS, G. and J.D. PAYNE. 1981. Time course of photoreactivation of UV-induced chromosomal aberrations and lethal damage in interphase *Xenopus* cells. Photochem. and Photobiol. 35:57-62.

HARM, W. 1980. Biological effects of ultraviolet radiation. Cambridge University Press, London, pp. 23-39.

KULP, S., A. PAGE, and G. GRIGGS. 1985. Repair of lethal damage induced in a hamster x amphibian hybrid tissue culture cell line by ultraviolet light and gamma-ray. Proc. Ark. Acad. Sci. 69:69-73.

WOLFF, S. 1961. Radiation genetics. Mechanisms in radiobiology. Academic Press, New York. 1:419-475.

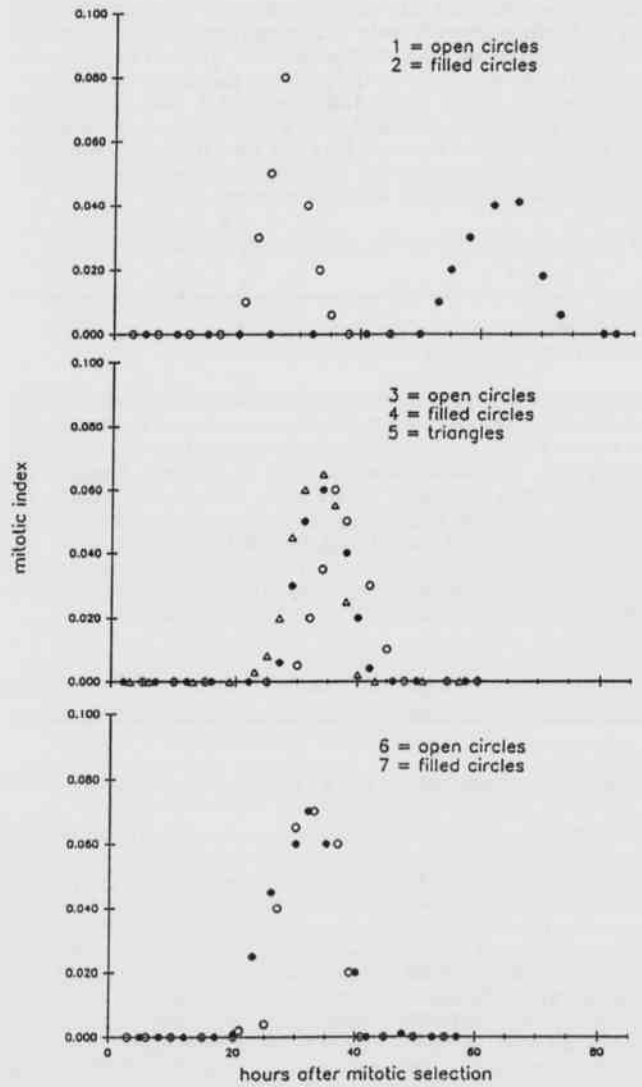

Figure 1. Mitotic index curves determined with sets of A8W4 cells, which were exposed to 200 rads gamma ray in early G1 phase (one hour after mitotic selection) and then exposed to varying UV and PR light fluences as follows: (1) 0.0 J/m² UV and 0.0 J/m² PR light, control; (2) 8.0 J/m² UV and 0.0 J/m² PR light; (3) 8.0 J/m² UV and 20.0 x 10^3 J/m² PR light; (4) 8.0 J/m² UV and 23.0 x 10^3 J/m² PR; (5) 8.0 J/m² UV and 26.0 x 10^3 J/m² PR light; (6) 8.0 J/m² UV and 30.0 x 10^3 J/m² PR light; (7) 8.0 J/m² UV and 35.0 x 10^3 J/m² PR light. These curves, 1 through 7, are the MI curves for experiments 1 through 7 (of the table), respectively. For each curve the onset of the UV exposure was immediately following the termination of the gamma ray exposure and the onset of the PR exposure was immediately following the termination of the UV exposure. The mitotic index of the cultures of mitotic selection was approximately 0.98.

Table 1. Photoreactivation of the effect of UV on gamma ray-induced aberration production in A8W4 cells. In each experiment synchronous cultures of G1 phase cells were first exposed to 200 rads gamma ray (one hour after mitotic selection) and then exposed to UV and PR fluences as indicated.

Experiment Number	UV fluence (J/m^2)	PR fluence ($J/m^2/10^3$)	Cell collection time range (hours after mitotic selection)*	Chromosome type aberrations**	
				Deletions	Exchanges
1	0.0	0.0	23-35	105	66
2	8.0	0.0	54-70	164	43
3	8.0	20.0	30-45	144	48
4	8.0	23.0	28-40	140	51
5	8.0	26.0	28-40	141	52
6	8.0	30.0	26-40	142	49
7	8.0	35.0	26-40	139	50

* Cells were collected for aberrational analysis by colcemid treatments that spanned the indicated time ranges.

** 300 cells were scored in each experiment.

REBECCA ROWE and GASTON GRIGGS, Department of Biology, John Brown University, Siloam Springs, AR 72761.