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SAGE THRASHER *(OREOSCOPTES MONTANUS)*, A NEW STATE RECORD

On 24 November 1979, Cheryl Lavers and I were looking for birds in the Farville area, about 6 miles NE of Jonesboro, Craighead County, when we discovered a Sage Thrasher *(Oreoscoptes montanus)*. The bird was rather Mockingbird-like *(Mimus polyglottos)* in shape, plain gray-brown crown and back, and bright yellow eye with a black pupil. Other characters differed from a Mockingbird in that it was smaller and shorter tailed and it had a white throat with a black malar stripe. The underparts, with a ground color of warm buff, were densely streaked with black chevrons arranged in length-wise rows. There was a white wingbar, and a dark tail with white outer corners. The bill was slender, very slightly de-curved, and dark bluish-black in color, as were the rather short legs. The bird ran along the ground in open places, or under brush and stayed on or near the ground. It was observed catching, decapitating, then swallowing ground crickets (*Acheta* sp.). We have both observed Sage Thrashers in several western states. An examination of standard field guides (Peterson, R. T. 1961. A field guide to western birds. Houghton Mifflin Co., Boston; Robbins, C. S. et al. 1966. Birds of North America. Golden Press, N.Y.) further confirmed our identification. A description and slides of the bird have been sent to Charles Mills, Curator, the Arkansas Audubon Society, and Dr. Douglas James, Department of Zoology, University of Arkansas at Fayetteville. This is a first recorded instance of the Sage Thrasher in Arkansas.

In its normal range, the Sage Thrasher breeds in the sagebrush *(Artemisia)* deserts of western North America, coming as far east as western Oklahoma. It winters in the southern part of its range and Mexico, occurring as far east as central and southern Texas, with a small isolated winter colony in extreme southwestern Louisiana (A.O.U. check-list of North American birds, 1957). The species is seldom recorded as a vagrant. The following extralimital occurrences to the east of its range have been recorded: Florida, 2; Illinois, 2; Maryland, 1; New York, 2; North Carolina, 1; South Dakota, 1.

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SOME EFFECTS OF METHYL GREEN ON EXPRESSION OF DAMAGE INDUCED IN G1 *XENOPUS* CELLS BY ULTRAVIOLET LIGHT

Methyl green shows a high degree of specificity for DNA and is a component of many nuclear stains. Kurnick (1952) observed that this basic dye was readily bound by polymerized DNA; two amino groups of the dye binding to two phosphoric acid groups of DNA. Errera (1951) demonstrated that the affinity of DNA for methyl green is influenced by radiation and other agents which depolymerize DNA, or alter its internal configuration. Doudney and Haas (1958) showed that methyl green significantly influenced metabolic activities, such as DNA and RNA synthesis, in *Escherichia coli.* These results suggested that appropriate experiments, involving methyl green treatments coupled with germicidal UV exposures, might aid in describing the expression and repair of UV-induced lethal and mutational damage in prokaryotic cells. Experiments of this nature were carried out by investigators, such as Witkins (1961), which led to the notion that UV induces lesions in bacterial DNA that either are removed by repair systems or are converted to permanent structural changes during the first DNA synthetic period following the exposure. We report here an extension of such experiments to eukaryotic cells, in which some effects of methyl green on the repair of UV (254 nm)-induced damage in G1 phase *Xenopus* cells are examined.

Routine maintenance of log phase A8W243 *Xenopus* cultures, incubations, cell synchronizations, irradiations, mitotic index determinations, survival determinations (colony counts), and chromosome analysis have been described in detail (Griggs and Bender, 1973; Payne and Griggs, 1977; Griggs and Orr, 1979).

The basic dye used, methyl green, was obtained from Difco.

Figure 1 shows results of a series of mitotic index experiments performed to examine the effects of methyl green on progression of UV irradiated G1 cells through interphase and the first succeeding mitosis, (M1). These data described the appropriate time intervals for collection of the sets of mitotic cells analyzed for effects of methyl green on UV-induced aberration production (Table 1). Concentrations of the dye in the range 0.0 - 0.003 g/1 appeared to induce little delay in progression of the irradiated cells above that induced by the UV alone. The similarity in average height and width of these mitotic peaks indicated that the dye did not significantly reduce the number of irradiated cells that reached G1.

The average cell cycle for non-irradiated *Xenopus* cells was approximately 26 hours (not shown); eight hours G1, 13 hours S, three hours G2, and two hours M1. Payne and Griggs (1977) carried out autoradiographic studies which indicated that early G1 phase cells, exposed to low doses of UV (0 - 150 ergs mm^{-2} range), are not delayed in their progression through G1, but experience rather lengthy S phase delays. These facts, coupled with the data of Figure 1 and Table 1, indicate that chromatid aberration frequencies, resulting from UV exposure of early G1 cells, are significantly altered by methyl green only when the dye is in contact with the exposed cells as they pass through early S phase. Some relationship between the aberrant intracellular mechanism, by which methyl green augments chromatid type aberration production, and DNA synthesis is suggested by the fact that both mechanisms appear to function with peak efficiency in early S phase.

The data of Figure 2 indicate that a methyl green concentration of 0.003 g/1 has virtually no effect on the expression of UV-induced lethal damage, no matter where in the cell cycle the dye is applied. These data suggest that the mechanism which expresses UV-induced aberrational damage in *Xenopus* cells differs significantly from the mechanism which expresses lethal damage, supporting results of previous studies of the overlap of UV-induced lethal and aberrational lesions in *Xenopus* cells (Griggs and Orr, 1979; Payne and Griggs, 1977).