of environmental changes in this area, the shipboard scientific party has refrained from sampling any more than the core catcher samples in order to preserve the cores for varve studies onshore, which require intact working halves of the cores. We proposed to the DSDP and the National Science Foundation that these cores be curated and samples distributed in a special way. A HPC varved core working group has been established within the Joint Oceanographic Institutions for Deep Earth Sampling, which will coordinate feasible ways to subsample the working half cores for subsequent shore-based studies.

HANS SCHRADER
School of Oceanography, Oregon State University, Corvallis 97331
KERRY KELTS, JOSEPH CURRAY
DAVID MOORE
Scripps Institution of Oceanography, La Jolla, California 92037
EDUARDO AGUAYO
Instituto Mexicano del Petroleo, México 14, D.F.
MARIE-PIERRE AUBRY
Woods Hole Oceanographic Institution, Woods Hole, Massachusetts 02543
GERHARDT EINSELE
Geologisches Institut, 74 Tubingen, Federal Republic of Germany
DANIEL FORNARI
Lamont-Doherty Geological Observatory, Palisades, New York 10964
JORIS GIESKES
Scripps Institution of Oceanography
JOSE GUERRERO
Universidad Nacional Autónoma de México, México 20, D.F.
MIRIAM KASTNER
Scripps Institution of Oceanography
MITCHELL LYLE
School of Oceanography, Oregon State University
YASUMOCHI MATOBA
Department of Geology, Stanford University, Stanford, California 94305
ADOLFO MOLINA-CRUZ
Centro de Investigación Cientifica y de Educación Superior de Ensenada, Oceanografia, Ensenada, Baja California, México
JEFFREY NIEMITZ
Department of Geology, Dickinson College, Carlisle, Pennsylvania 17013
JAIME RUEDA
Instituto Mexicano del Petroleo
ANDREW SAUNDERS
Department of Geology, Bedford College, London NW1 4NS, England
BERND SIMONEIT
University of California, Los Angeles 90024
VICTOR VAQUIER
Scripps Institution of Oceanography

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7. The HPC operates on the principle of a 4.45-m core barrel which is lowered inside the drill string, hydraulically ejected into the sediment, and retrieved. The pipe is then lowered those 4.45 m to the next interval, and the procedure is repeated. At site 480 we pulled 31 cores (Fig. 2) and obtained about 118 m or 80 percent of the 152-m section with two cores empty and one core washed. Because of a misconception, the drill string was lowered one-half stand for each core or 4.75 m rather than the 4.45 m; this procedure produced even greater gaps in the record, although the recovery success of the HCP then increases to over 86 percent. The top and bottom approximately 20 cm of a core generally showed some sediment disturbance, but most of the sections survived undisturbed with more than 76 m consisting of finely laminated oozes. The ocean was becalmed at site 480, so there is no uncertainty in a core's exact depth position resulting from the ship's heave, although tidal effects are not compensated. The site is located at 27°54'N, 111°39.3'W in 655 m of water [J. Curray *et al.*, *Geotimes* **24**, 18 (1979)].
8. The number of dark-light couplets per centimeter is uniform within error throughout the section. Random counts of laminae reveal 12 to 15 couplets per centimeter in the top four cores and 12 to 29 couplets per centimeter in the bottom four cores. This slight increase in the frequency as well as a slight thinning of laminae suggests compaction of the sediment rather than an increase in the number of couplets deposited per unit time.
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Fluorescent Light Induces Malignant Transformation in Mouse Embryo Cell Cultures

Abstract. *Fluorescent light induced a dose-dependent malignant transformation in mouse C3H10T$^1/_2$ cells. A plateau in the dose-response curve for transformation was correlated with that observed with ultraviolet light exposure. The similarity in the two dose-response patterns suggests that similar molecular processes may be involved in the induction of malignant transformation by the two types of radiation.*

Light from standard fluorescent bulbs is toxic and mutagenic to bacteria (*1*) and to mammalian cells in vitro (*2-5*). Fluorescent light also induces breaks in DNA strands (*6*), cross-links, and chromosomal aberrations (*7*) in cultured cells, as well as sites sensitive to *Micrococcus luteus* endonuclease (*8*). These sites are thought to represent pyrimidine dimers, a DNA lesion previously implicated in cancer induction (*9, 10*) and in vitro malignant transformation (*11*). We report here that fluorescent light is capable of transforming cells in vitro and that the frequency of malignant transformation induced is related to dose.

We used a cell line derived from mouse embryo (C3H10T$^1/_2$, clone 8) isolated and characterized by Reznikoff *et al.* (*12, 13*) and adapted for studies of radiation transformation in our laboratory (*14*). The cells were passaged and maintained as previously described (*14, 15*). Cultures containing about 2×10^6 cells in uncovered 100-mm petri dishes were irradiated with six fluorescent bulbs (GE F15-T8 Cool White) at a distance of 6 inches. The exposure rate was 27.3 J m^{-2} sec^{-1}, measured with a thermopile (Eppley). This was equivalent to 910 foot-candles (1 foot-candle = 10.76 lux) (International Light IL700 meter). All irradiated and control (exposed only to dark) dishes were maintained at 0° to 4°C in Hanks balanced salt solution (HBSS) containing 25 m*M* Hepes buffer (*p*H 7.1, HBSS) (*16*), during exposure periods ranging from 0 to 7 hours. The plates were then subcultured to 250 to 400 viable cells per plate and maintained for radiation transformation (*14*). Types II and III foci were scored separately as transformants; their morphology was similar to that previously observed for ultraviolet light (*11*), x-irradiation (*14*), and chemical carcinogen (*12, 13*) treatments. There was no spontaneous transformation in control cultures (no treatment) or in cultures maintained at 37°C throughout but incubated for 7 hours in HBSS.

Figure 1 (upper curves) presents survival data for 0 to 7 hours (0 to 6.9×10^5 J m^{-2}) exposure to fluorescent light, as well as for cultures kept in the dark at 0°

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Table 1. Fluorescent light induced malignant transformation. For the transformation assay, 100-mm plastic dishes (Falcon) were seeded with 250 to 400 surviving cells after plates containing cells in exponential growth were exposed to fluorescent light. The transformation data have been pooled from three to four separate experiments of 20 to 60 plates each for each exposure time. Plating efficiencies were determined from 3 to 6 plates seeded with a cell density one-fifth that of the plates for the transformation assay; cultures were terminated 10 days after the fluorescent light exposure.

Treatment (hours)	Dark controls			Irradiated		
	Transformed foci*/plate		Transformation frequency per surviving cell, types II and III ($\times 10^{-4}$)	Transformed foci*/plate		Transformation frequency per surviving cell, types II and III ($\times 10^{-4}$)
	Type III	Types II and III		Type III	Types II and III	
2	0/74	0/74	< 0.4	0/77	2/77	0.9
$2^1/_2$	0/38	0/38	< 1.6	1/73	4/73	2.0
3	0/37	0/37	< 1.6	2/76	8/76	3.3
$3^1/_2$	0/72	1/72	0.4	1/70	9/70	4.7
5	0/55	1/55	0.6	1/60	10/60	4.1
7	0/147	2/147	0.5	3/152	8/152	3.7
5 (with petri dish covers on)				0/34	0/34	< 1.8

*Or number of plates containing transformed foci.

to 4°C for the same periods of time. The frequencies of malignant transformation induced by similar doses of fluorescent light are shown in Table 1. The transformation frequency increased with increasing light exposure from 0 to $3^1/_2$ hours (0 to 3.4×10^5 J m^{-2}) and reached a plateau at doses greater than or equal to 3 to $3^1/_2$ hours. Exposure to fluorescent light for 5 hours with covers on the petri dishes resulted in no detectable transformation.

Exposure of control cultures to 0° to 4°C in the dark resulted in the killing and malignant transformation of some cells (Fig. 1 and Table 1); however, no type III foci were observed. Transformation in nonirradiated cultures cannot be attributed to the 2- to 7-hour incubation with HBSS, as parallel control cultures maintained at 37°C for similar times in this medium showed no transformation. Malignant transformation induced by x-rays is enhanced when cultures are irradiated and maintained for 4 hours thereafter at 5°C rather than at 37°C (*15*). It is possible that exposure from 2 to 7 hours to 0° to 4°C increases the spontaneous transformation frequency by a similar mechanism. Light exposure, however, increased the transformation frequency by about tenfold over these control levels (Table 1).

The transformation data for fluorescent light exposure from Table 1 are plotted in Fig. 1. Also included is the transformation dose response curve reported by Chan and Little (*11*) for germicidal (254-nm) ultraviolet light. The experimental conditions we used are the same as those previously used to demonstrate that fluorescent light induces DNA sites sensitive to *M. luteus* endonuclease (*8*). Fluorescent light doses may be related to equivalent ultraviolet light doses in terms of numbers of endonuclease-sensitive sites induced by each treatment. One hour of fluorescent light exposure under our experimental conditions produces as many sites as 2.7 J m^{-2} of germicidal ultraviolet light exposure (*17*). The ultraviolet light and fluorescent light scales (Fig. 1) have thus been adjusted to represent equal numbers of sites sensitive to endonuclease. The observed plateau in the curve of transformation frequency versus fluorescent light dose correlates well with the results for the transformation induced by ultraviolet light. The similarity of these two patterns suggests that similar molecular processes may be involved in the induction of malignant transformation by the two types of radiation.

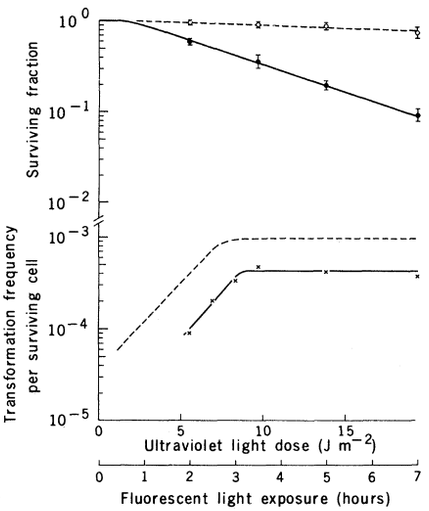

Fig. 1. (Upper curves) Survival data for fluorescent light exposures. Each point represents the mean ± standard error for three to five separate experiments per point, each involving three to six dishes. Open circles represent survival of control cultures kept in the dark at 0° to 4°C for the exposure periods shown. Filled circles represent survival for irradiated cultures maintained under the same conditions. (Lower curves) Transformation frequency per surviving cell as a function of fluorescent light dose. The dashed line represents transformation frequency as a function of germicidal (254-nm) ultraviolet light dose (*11*). Fluorescent light exposure is measured in hours and is plotted in direct relation to the ultraviolet dose in terms of their equivalence in the number of sites sensitive to endonuclease induced in the cellular DNA. Data points for 3 to $3^1/_2$ hours to 7 hours are drawn as a plateau, as no statistically significant difference can be demonstrated with a χ^2 test of the data in Table 1, column 6.

Sites induced by ultraviolet light that are sensitive to *M. luteus* endonuclease are pyrimidine dimers (*18*), and the majority of sites induced by fluorescent light are probably also pyrimidine dimers. This is to be expected because fluorescent lamps emit about 2 percent of their output at light wavelengths between 300 and 400 nm. Such wavelengths induce dimers at low but measurable rates (*19*). We confirmed the specificity of our endonuclease preparation for pyrimidine dimers as follows: (i) Results obtained with our *M. luteus* endonuclease preparation [a crude extract equivalent to fraction II of Carrier and Setlow (*20*)] were compared with those obtained when a highly purified, dimer-specific preparation (*18*) was used (a gift of L. Grossman). These two preparations gave identical results for both germicidal ultraviolet light and fluorescent light exposures. (ii) One-dimensional thin-layer chromatography (*21*) was used to identify and measure pyrimidine dimers induced by fluorescent light. Parallel chromatographic and endonuclease site measurements of DNA from cells exposed to 8 hours of fluorescent light yielded 0.11 ± 0.02 percent of thymine bases in dimers, equivalent to 62.5 ± 10.4 dimers per 10^8 daltons of DNA (*22*), and yielded 53.2 ± 4.6 endonuclease sites per 10^8 daltons, respectively. Thus, a given dose of fluorescent light

induces similar numbers of pyrimidine dimers and endonuclease sites. These results suggest that the endonuclease assay used in this study exclusively detects pyrimidine dimers in DNA damaged by fluorescent light. Thus, the equivalent patterns seen when fluorescent light and ultraviolet light transformation are plotted against numbers of endonuclease sites (Fig. 1) suggest that the pyrimidine dimer is the common causal agent in transformation by these radiations. The experiments were performed at 0° to 4°C to minimize the potential for repair of these dimers during the long exposure times.

The transformation frequencies observed for fluorescent light in the plateau region of the dose-response curve (Fig. 1) are somewhat lower than those observed by Chan and Little (*11*) for germicidal ultraviolet exposure. Although the reason for this lower apparent efficiency of fluorescent light is unclear, the fact that the transformation frequencies were not higher than those observed for ultraviolet light suggests that base damage by fluorescent light (other than pyrimidine dimers) may not be effective in inducing transformation.

The involvement of other DNA photoproducts, however, cannot be ruled out. For example, thymine glycols (monomeric ring-saturated products of the 5,6-dihydroxydihydrothymine type) are a minor DNA photoproduct relative to dimers after irradiation at 254 nm, but become a numerically important product at 313 nm (*23*). The biological importance of glycols is at present unclear, but these lesions might be involved in the fluorescent light effects seen here. Other DNA photoproducts are induced by near ultraviolet and visible light through photodynamic action, a mechanism involving the interaction of light with certain fluorescent, cellular components (*24*). Such damage has been implicated in bacterial mutagenesis (*25, 26*) and could also be a causal agent in transformation by fluorescent light. In addition, DNA strand breaks (*6*) and DNA cross-links (*7*) are induced by fluorescent light. Thus, the involvement of nondimer damage in the transformation we have observed cannot be excluded.

Normal tissue culture conditions should reduce fluorescent light effects. For example, no detectable transformation was observed after 5 hours of exposure through an intervening plastic dish cover (Table 1); presumably the plastic blocked the transmission of a small component of more biologically effective, shorter wavelength in the fluorescent light emission spectrum. The use of gold fluorescent lights would probably also reduce fluorescent light transformation; commercial gold lights, which do not emit wavelengths below 5000 Å, are not mutagenic to mammalian cells (*4*).

Fluorescent light should be regarded as a perturbing but controllable (by glass or plastic filtration) agent in all in vitro survival, mutagenesis, and malignant transformation experiments. The results also suggest that fluorescent light exposure could contribute on a small scale to human skin carcinogenesis.

ANN R. KENNEDY
Laboratory of Radiobiology, Harvard School of Public Health, Boston, Massachusetts 02115
MARK A. RITTER
Department of Radiation Therapy, School of Medicine, University of Pennsylvania, Philadelphia 19104
JOHN B. LITTLE
Laboratory of Radiobiology, Harvard School of Public Health

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The Oaxaca, Mexico, Earthquake of 29 November 1978: A Preliminary Report on Aftershocks

Abstract. *Aftershocks of the 29 November 1978 Oaxaca, Mexico, earthquake (surface-wave magnitude* $M_s = 7.8$*) define a rupture area of about 6000 square kilometers along the boundary of the Cocos sea-plate subduction. This area had not ruptured in a large* ($M_s \geq 7$)*, shallow earthquake since the years 1928 and 1931 and had been designated a seismic "gap." The region has also been seismically quiet for small to moderate* ($M \geq 4$)*, shallow (depth* ≤ 60 *kilometers) earthquakes since 1966; this quiet zone became about six times larger in 1973. A major earthquake* ($M_s = 7.5 \pm 0.25$) *was forecast at this location on the basis of the quiescence that began in 1973. The aftershock data indicate that an area approximately equivalent in size to the seismic gap has now broken.*

On 29 November 1978, a large (surface-wave magnitude $M_s = 7.8$) earthquake ruptured the Middle America trench along the coast of Oaxaca, Mexico (Fig. 1). This earthquake attracted particular attention because both its location and size had been forecast by seismologists. The specific time of occurrence, however, was not forecast (*1, 2*).

The seismic potential of this location, first pointed out in 1973, was based on recurrence rates of 50 years or less for large ($M_s \geq 7.0$), shallow (depth ≥ 60 km) earthquakes in Mexico and Central America (*1*). Rupture had last occurred in 1928 and 1931, and the area was designated a seismic "gap." The dimensions of the gap were subsequently used to estimate the magnitude ($M_s = 7.5 \pm 0.25$) of a future earthquake (*2*). It was also suggested (*2*) that small to moderate earthquakes ($M \geq 4$) should cease and then resume prior to the main shock in a

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