- ¹⁶ Folch-Pi, J., and Sherman, G., Second Intern. Meeting Intern. Soc. Neurochem., 169 (Milan, 1969).
- Soc. Neurochem., 169 (Milan, 1969).
 Durell, J., and Garland, J. T., Ann. NY Acad. Sci., 165, 743 (1969).
 Clayton, P. A., and Rowe, C. E., Biochem. J., 101, 674 (1966).
 Lunt, G. G., and Lapetina, E. G., Brain Res., 17, 164 (1970).
 González-Rodríguez, J., La Torre, J. L., and De Robertis, E., Mol. Pharmacol., 70, 122 (1970).
 De Robertis, E., González-Rodríguez, J., and Teller, D. N., FEBS Lett., 4, 4 (1969).
 Vicenez, C. Barrantes, E. L. La Torre, L. L. and De Robertis.
- ²² Vásquez, C., Barrantes, F. J., La Torre, J. L., and De Robertis, E., *J. Mol. Biol.*, 52, 221 (1970).
- Eichberg, J., Whittaker, V. P., and Dawson, R. M. C., *Biochem. J.*, 92, 91 (1964).
- ²⁴ Durell, J., Garland, J. T., and Friedel, R. O., Science, 165, 862 (1969).

- 862 (1969).
 Hokin, M. R., J. Neurochem., 16, 127 (1969).
 Lunt, G. G., and Lapetina, E. G., Brain Res., 18, 451 (1970).
 Hokin, M. R., and Brown, D. F., J. Neurochem., 16, 475 (1969).
 Folch, J., Lees, M., and Sloane-Stanley, G. H., J. Biol. Chem., 226, 497 (1967).
 Neskovic, N. M., and Kostic, D. M., J. Chromatog., 35, 297 (1968).
 Chen, P. S., Toribara, T. Y., and Warner, H., Analyt. Chem., 28, 1756 (1956) 28, 1756 (1956).

Susceptibility of Trisomic and of Triploid Human Fibroblasts to Simian Virus 40 (SV40)

CELLS from patients with G-trisomy or E-trisomy and XXY cells from patients with XY/XXY mosaic Klinefelter's syndrome

Table 1 Induction of SV40 T Antigen in Human Fibroblast Strains

| | Cell | | % cells with T antigen Individual Group | |
|------------|---|-------------------------------------|---|---------|
| Group | Strain | Passage | test | average |
| Normal | CR CR CR CR CR HALL SUL | 9 13 16 18 19 2 6 | 8.5 5.8 11.4 9.0 9.0 12.1 5.9 | 8.8 |
| Triploid | PER PER PER PER | 2 5 7 11 | 3.8 6.6 10.6 10.3 | 7.8 |
| G-Trisomic | KIN KIN KIN GAL | 6 8 19 9 | 21.7 17.7 17.2 15.8 | 18.1 |
| Fanconi | FAN FAN | 1 5 | 41.6 37.9 | 39.8 |

A single pool of SV40, strain 776 (ref. 6), with a titre of 2.0×10^8 plaque forming units in BSC-1 cells was used. All fibroblast cultures were derived from explants of skin biopsies and grown in modified⁸ Eagle's minimal essential medium. Cultures were incubated at 35° C in an atmosphere of 5% CO₂ and air. For infection, cultures were initiated with 3.5 × 10⁵ cells in 5 ml. of medium per 2 ounce prescription bottle. Two days later the medium was renewed. On the third day, the medium was removed, the cells were washed twice with 2 ml. of Hanks balanced salt solution and 0.5 ml. of undiluted SV40 suspension was added. After a 4 h viral adsorption period, the cells were again washed twice and fresh medium added. Unlike previous investigators^{2,5} we did not maintain the infected cultures in anti-SV40 serum. Preliminary experiments showed that the relative susceptibilities of several cell strains to SV40 T antigen induction were not altered by maintenance of the infected cultures in medium containing 1.0% monkey anti-SV40 serum (95% plaque reduction titre=1:10,000). For each cell strain tested, maintenance in anti-SV40 serum halved the percentage of cells producing T antigen at 72 h after infection. 18 h after infection, the cells were treated with trypsin and suspended in medium to give 5 × 10⁴ cells/ml. One ml. of cell suspension was distributed to each of 3 Leighton tubes containing coverslips. At 72 h after infection the cells were fixed in acetone. T antigen was detected by the indirect immunofluorescent technique using serum from hamsters bearing SV40-induced tumours9. The percentage of cells containing T antigen was estimated from counts of 1,500 cells.

are more susceptible to transformation in vitro by SV40 than are cells from normal individuals¹⁻³. We have used triploid (69XXY) human cells to determine whether the presence of extra chromosomes per se increases susceptibility to transformation.

The susceptibility of a human fibroblast strain to SV40induced transformation is given by the proportion of cells which, when in dividing cell culture, produces SV40 T antigen at 72 h after exposure to the virus^{2,5}. In addition to cells from two patients with G-trisomy and the patient with triploidy. cells from three clinically normal individuals and a patient with Fanconi's anaemia were included as controls.

Table 1 shows that, on average, 18.1% of trisomic cells and 7.8% of triploid cells produced T antigen. This latter figure is very similar to that of normal diploid cells. For cells from patients with G-trisomy and from the patient with Fanconi's anaemia the relatively high percentage of cells which produced T antigen is in accord with other reports^{1,2,5,10} that such cells are unusually susceptible to SV40.

That the susceptibility of the triploid cells to SV40 is essentially normal indicates that the increased susceptibility of trisomic cells, at least as judged by T antigen production, results from factors other than the simple presence of the extra chromosome. The chromosomal imbalance present in the trisomic cells but not in the triploid cells is probably involved in the increased susceptibility to SV40.

This study was supported in part by a US Public Health Service research grant from the National Cancer Institute. We thank Karen Findling Day, Patricia J. Carmody and Jan Pooley for technical assistance.

FRANCIS E. PAYNE

Virus Laboratory. Department of Epidemiology, School of Public Health

ROY D. SCHMICKEL

Department of Pediatrics, School of Medicine, University of Michigan, Ann Arbor, Michigan 48104

Received October 28, 1970.

- ¹ Todaro, G. J., and Martin, G. M., Proc. Soc. Exp. Biol. Med.,
- 124, 1232 (1967).

 Potter, C. W., Potter, A. M., and Oxford, J. S., J. Virology, 5, 293 (1970).
- ³ Mukerjee, D., Bo 30, 1769 (1970). Bowen, J., and Anderson, D. E., Cancer Res.,
- 30, 1769 (1970).

 Schmickel, R. D., Silverman, E. M., Floyd, A. D., Payne, F. E., Pooley, J. M., and Beck, M. L., J. Paediatrics (in the press).

 Aaronson, S. A., and Todaro, G. J., Virology, 36, 254 (1968).

 Meyer, jun., H. M., Hopps, H. E., Rogers, N. G., Brooks, B. E., Bernheim, B. C., Jones, W. P., Nisalak, A., and Douglas, R. D., J. Immunol., 88, 796 (1962).
- ⁷ Hopps, H. E., Bernheim, B. C., Nisalak, A., Tijo, J. H., and Smadel, J. E., *J. Immunol.*, 91, 416 (1963).
- Krooth, R. S., and Weinberg, A. N., J. Exp. Med., 113, 1155
- Bisset, M. L., and Payne, F. E., J. Bact., 91, 743 (1966).
 Todaro, G. J., Green, H., and Swift, M. R., Science, 153, 1252
- (1966).

Acceleration of Senescence induced in Wheat by Light

THE effects of low light intensity which rapidly lead to observable changes in the growth rate and morphology of plants are well documented1. In an experiment to determine the influence of low intensity light on the yield of grain in wheat, we have discovered a new effect which is expressed several weeks after treatment.