Table 1. GAPS, BREAKS AND HYPERDIPLOID CELLS IN PATIENTS TREATED WITH LSD OR PSYCHOPHARMACOTROPHIC DRUGS, AND IN CONTROLS

	Mean age and $S.D.$	Cells examined	Gaps Total Per cent		Breaks Total Per cent		Hyperdiploid cells Total Per cent	
Five psychiatric patients treated with LSD Seventeen psychiatric patients treated with	$36{\cdot}4\pm6{\cdot}47$	358	57	15.9	6	1.7	7	2.0
psychotropic drugs Thirteen psychiatric patients given no drugs Ten hospital staff members	$\begin{array}{c} 36 \cdot 9 \pm 8 \cdot 89 \\ 32 \cdot 8 \pm 6 \cdot 28 \\ 27 \cdot 8 \pm 5 \cdot 04 \end{array}$	510 476 326	$67 \\ 33 \\ 18$	$13.1 \\ 6.9 \\ 5.5$	5 0 0	$ \begin{array}{c} 1 \cdot 0 \\ 0 \\ 0 \end{array} $	1 0 0	0·2 0 0

with drugs and those not treated with drugs, we found that the frequency of gaps and breaks in the group treated with drugs was significantly higher than in the group of patients not treated with drugs ($\chi^2 = 10.399$, P < 0.005, P(Fisher) = 0.037, Table 1).

The difference between the frequency of gaps in the group of patients treated with drugs and the staff members group was significant ($\chi^2 = 12.629$, P < 0.0005), but the difference between the frequency of breaks in the two groups was not significant (P(Fisher) = 0.084).

This indicates that some of the psychotropic drugs may induce gaps and breaks to the extent found in patients We found, however, a significantly treated with LSD. higher frequency of hyperdiploid cells in the patients treated with LSD than in the control patients treated with psychotropic drugs (P(Fisher) = 0.010). There was no significant correlation between any specific drug and the frequency of gaps, breaks and hyperdiploid cells in our comparatively small amount of material.

Further work is needed on the toxicity of different psychotropic drugs and other drugs. Any new drug should be tested for toxic effect on chromosomes, in animals and in vitro, before they are released for general use.

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Results of Consecutive Matings of Female Anopheles gambiae Species B with Fertile and Sterile Males

THE Anopheles gambiae complex consists of five closely related species; two salt water ones, A. melas and A. merus, and three fresh water ones, provisionally designated A, B and C. When males from species A or B are crossed with females of A. melas or A. merus the F_1 generation consists almost entirely of sterile males. It has been proposed that the release of such sterile males be used to control members of this species complex¹. It has already been established that these males compete successfully against normal males when both are caged together with virgin females1. I have carried out experiments to investigate whether a female, once mated with a sterile male, could lay fertile eggs if she subsequently mated with a fertile male, or if a female, once mated with a fertile male, could be sterilized by mating with a sterile male. Previous authors, seeking evidence of multiple insemination in Anopheles spp. with the aid of marker genes, found very little indication of it^{2,3}.

Mosquitoes used were females and fertile males from a species B colony from Kano, Nigeria, and sterile males produced by crossing the Kano males with Anopheles melas females from a strain from Liberia. Fifty-six females, which had been caged with fertile males and had laid fertile eggs, were re-fed, hand-mated⁴ each to a separate sterile male, and allowed to lay again. In fiftyfour cases, the succeeding batches of eggs were again fertile. In only two cases did the eggs fail to hatch. Seventy-five females caged with sterile males laid eggs which failed to hatch. Virgin females of species B seldom oviposit¹ and it can be assumed that most of these females had mated with the sterile males. On re-feeding, and after being hand-mated to fertile males, all seventy-five females again laid eggs which failed to hatch. Thirty of these females oviposited again, twelve of these laid a fourth batch of eggs and two laid a fifth batch. All were sterile.

These results are in agreement with those of George⁵, who found in Aedes aegypti that the first mating is the most important.

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Dark Reactivation of Damage induced by Ultraviolet Light in Mammalian Cells in vitro

INDUCTION of pyrimidine dimers in cellular DNA by irradiation with ultraviolet light and the molecular processes involved in the repair of this damage (photoreactivation¹ and dark reactivation²⁻³) are established in bacteria, bacteriophage and transforming DNA. Investigations of the reactivation of ultraviolet-induced damage have been extended from micro-organisms to cover cultured mammalian cell lines4-6.

We wished to determine the difference in sensitivities of cultured mammalian cell lines to X-rays and ultraviolet light to find out whether the reactivation processes after damage by X-rays and ultraviolet light are identical, and to see whether mechanisms can be demonstrated which split or excise ultraviolet-induced thymine dimers. Mouse fibroblastic L cells, porcine kidney stable (PS) cells, and mouse Ehrlich ascites tumour cells maintained in vitro were used.

For determinations of colony forming ability, cells of each line were plated in 60 mm glass Petri dishes containing 70 per cent TC-199 medium plus 10 per cent 'Bacto' tryptose phosphate broth (Difco) supplemented with 20 per cent bovine serum. The dishes were incubated at 37° C for 10 to 14 days in a humidified CO₂ incubator which was flushed with a mixture of 5 per cent CO₂ and 95 per cent air. For X-irradiation, 5 ml. samples of cells were suspended in short 'Pyrex' test tubes (inner diameter 10 mm, length 100 mm), and irradiated with various doses at a rate in air of 64 r./min as measured with a Victoreen condenser chamber. After each 1 ml. of the unirradiated or irradiated cell suspensions had been distributed into 60 mm glass Petri dishes and supplemented with 4 ml. of culture medium, the dishes were placed in a CO₂ incubator. For ultraviolet irradiation, 1 ml. samples of cell suspensions were distributed into 60 mm glass Petri dishes, and irradiated with various doses at the predominant wavelength of 260 mµ from a 15 W germicidal lamp, at a dose rate of about 20 ergs/mm²/s at the surface of the dishes.