are induced with a frequency of about 20 per cent by exposures which give 46 per cent survival. Other mutations which reduce growth rate in the heterozygote occur with a frequency which is at least three times greater.

The effects of delayed death within clones produced by surviving cells are no doubt superimposed on the influences already mentioned. But it is clear that variation in clone size or growth rate cannot be used either as evidence of the existence of division probability or to calculate its parameters.

(*c*) Better evidence of the operation of division probability in yeast comes from pedigree analyses of surviving cells. The fact that irradiated cells produce clones which contain both dividing and non-dividing cells has been recognized for many years[8–10]. More recently, the phenomenon has been studied in detail[11,12]. In such studies, pedigrees are obtained by systematically separating the progeny of individual irradiated cells by means of a micromanipulator. This procedure reveals both the source and the pattern of production of non-dividing cells in mixed clones. Two pedigrees, which might be regarded as typical, are shown in Fig. 2. Such pedigrees are characteristic of more than half the survivors after exposures which give 50 per cent survival. Non-dividing cells appear thoroughout several generations as would be predicted by the theory, and the data emphasize a possible part played by chance in determining whether or not radiation damage is phenotypically expressed. Further, there is no doubt that radiation can produce a type of lethal damage in yeast which is not revealed by conventional plating procedures.

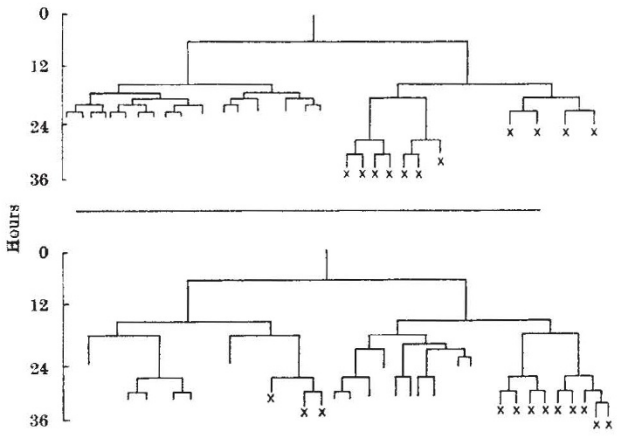

Fig. 2. Pedigrees of two X-irradiated cells of *Saccharomyces cerevisiae*. Each vertical line represents a separated cell, that on the left being the mother cell in any division. An *X* denotes that no further division occurred. Otherwise, reproduction continued with eventual production of a visible colony.

Nevertheless, the pattern that has emerged with accumulating data is not that expected of the concept. *a*, Non-dividing cells usually appear in clusters rather than as isolated cells. *b*, The size of a cluster varies from one to more than 100 cells. *c*, These clusters appear as sectors and seem to be a consequence of residual growth after the initiation of some lethal event in an earlier generation. *d*, The risk that a cell will be the progenitor of a lethal sector decreases in successive generations. Nevertheless, instability has been noted after as many as eight generations. *e*, The degree of instability is not uniform for all cells of an irradiated sample.

These characteristics do not exclude the existence of a rapidly shifting probability of non-division, but they rule out a constancy in the value of p_2 and in the number of generations between the occurrence of a lethal event and the termination of cell division. To be acceptable for yeast, the concept of division probability would have to be altered so drastically as to lose the practical usefulness claimed for it.

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"Congenital Anomalies" induced by X-Ray

IT was recently reported in your pages that prenatal X-irradiation produces a higher incidence of congenital anomalies in the offspring of primiparous mice than in those of multiparous mice, and that although the difference is not great, it is "indeed significant". In addition to gross external malformations, the classification "congenital anomaly" was also assigned by the writers to resorbed conceptuses, dead foetuses, and stunted survivors, although there is no justification for this practice, because these disparate phenomena may result from unlike effects of irradiation. Thus, collecting them together under one heading probably obscures rather than clarifies the nature of the damage done by prenatal irradiation.

I should like to go further and examine the authors' data[1]. When resorbed, dead and stunted offspring are omitted, it turns out that primiparous mice had 440 surviving young, of which 180 (40·9 per cent) were malformed; while the multiparae had 165/454 (36·3 per cent) malformed ones. This difference is indeed not great, but neither is it significant ($\chi^2 = 1{\cdot}78$, $P \cong 0{\cdot}2$).

It thus appears that the teratogenic susceptibility of the two groups of embryos was similar and that the previous reproductive history of the females was of no consequence.

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AGRICULTURE

Effect of Environment on Wheat Gliadin

CHEMICAL and physical investigations have shown that gluten composition differs from one wheat variety to another[1–5]. The total protein as a percentage of the weight of grain of a particular variety is affected by environmental factors and may be twice as great in some samples as in others grown in different conditions. Variations of this kind within a variety can result in an alteration in the proportions of the broad protein classes[6,7] and could alter the distribution of components within classes. The work reported here was designed to test as rigorously as possible whether the second kind of change occurs within the gliadin class of proteins. It is desirable to know whether or not this type of change can occur if information on the composition of the gliadin is to be readily applicable to genetic investigations of inheritance of wheat quality.