

the budding cycle, the non growing cells were added to fresh nutrient and shaken at 30°C. After 50–55 min incubation, the first small buds arose. The first budding cycle was completed by 125 to 130 min incubation.

Microscopic examination revealed that budding begins in good synchrony; 70–85 per cent of the cells showing small buds between 55 and 75 min. The rate at which the buds developed after their initial appearance was quite variable throughout the remainder of the cycle. However, since the DNA content of the cell doubles with the inception of budding¹, it was assumed that, regardless of variations in the size of buds during growth, an essentially synchronous series of nuclear changes was being examined.

For irradiation, cells were harvested after various periods of incubation, washed once in M/15 KH₂PO₄ and resuspended in M/15 KH₂PO₄ to a concentration of approximately 5 × 10⁵ cells/ml. Forty milliliters of the suspension contained in a sterile petri dish was irradiated at a rate of 1000 r/min with radiation emitted by a tungsten target tube operating at 180 KV/20 ma, 2 mm AL total filtration. Survival determinations were made by plating in a medium consisting of the broth described above solidified by the addition of 2 per cent agar.

Table

Time of Incubation	Target Values	Inactivation Rates
min		
0	3.5	0.101
55	1	0.037
75	1	0.027
90	1	0.035
120	1	0.042
135	1	0.042

Changes in the target values and inactivation rates over the course of a single budding cycle.

Control interphase cells yield sigmoid multitarget survival curves with an average target value of about 3.5 and a rate of logarithmic inactivation of 0.101 (Table). However, the survival curves of cells throughout the entire budding cycle are exponential (target value 1). At the inception of budding the rate of logarithmic inactivation decreases markedly with respect to that of the control. After 55 min incubation, the inactivation rate drops to 0.037 and after 75 min incubation drops still further to 0.027. By 90 min incubation, the inactivation rate rises to the value observed at 55 min and remains at that level until 120 min at which time it rises again to 0.042. Between 120 and 135 min incubation, when the second budding cycle is about to commence, no significant change occurs in the X-ray sensitivity of the cells.

These results agree well with the earlier studies by DARLINGTON and LACOUR² of the relation between chromosome breakage and the nucleic acid cycle. These workers demonstrated that the accretion of nucleotides by chromosomal structures rendered these elements resistant to breakage by X-rays. It is observed here that as nucleotides are produced during the early phases of

the budding process, the resistance of the yeast cell to X-ray inactivation greatly increases, and that as dechromatinization of the chromosomal structures occurs during the middle and later phases of the budding cycle the cellular resistance to inactivation decreases. The fact that the inactivation rate at the completion of the budding cycle does not fall to a value equal to that of the original interphase cells, together with the observation that the survival curve does not return to a sigmoid form, may reflect the fact that complete dechromatinization does not occur in cells of an actively dividing population.

These observations provide additional support for the earlier presumptive evidence¹ indicating that the inactivation of yeast cells by X-rays is in large part a consequence of the induction of chromosomal aberrations.

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Zusammenfassung

Es wurde die Beeinflussungsmöglichkeit der tetraploiden Hefezellen durch Röntgenstrahlungsinaktivierung während besonderer Stadien der Keimungsdauer untersucht. Das Kurvenbild der überlebenden, nichtkeimenden interphasen Zellen ist sigmaförmig und ergibt einen Zielwert 4 (target value 4). Die Kurvenbilder der Zellen sind während aller Phasen der Keimungsdauer exponential. Nichtkeimende Zellen zeigen der Inaktivierung gegenüber ein Mindestmass an Widerstand, Zellen im Mittelpunkt der Keimungsdauer ein Höchstmass an Widerstand. Aus diesen Versuchen darf man schliessen, dass eine Wechselbeziehung zwischen der Röntgenstrahlungssensibilität der Zellen und des Kernsäurezyklus während der Mitose besteht.

¹ A. SARACHEK, and W. H. LUCKE, Exper. 9, 374 (1953).

Der Einfluss der Pantothersäure, des Pantethins und des phosphorylierten Pantethins auf die Carotinbildung bei *Mucor hiemalis*

Die Pantothersäure ist als wesentlicher Bestandteil im Coenzym A eingebaut. Wie man weiss, spielt das Coenzym A bei den Acetylierungsreaktionen eine wichtige Rolle.

Unsere bisherigen Arbeiten über die Biosynthese der Carotinoide haben ergeben, dass die Essigsäure als eine primitive Vorstufe der Carotinoide anzusprechen ist¹. Somit stellt sich die Frage, ob das Coenzym A nicht auch die Bildung der Carotinoide beeinflussen würde, wie dies auch schon ARNAKI und STARY² angedeutet haben.

Über die ersten Ergebnisse der Experimente mit Pantothersäure, Pantethin und phosphoryliertem Pantethin, welche im Coenzym A (siehe Konstitutionsformel) eingebaut sind, soll hier kurz berichtet werden. Es standen uns Präparate von Pantethin und phosphoryliertem

¹ M. OGUR, S. MINCKLER, and D. O. McCLARY, J. Bact. 66, 642 (1953).

² C. D. DARLINGTON, and L. F. LACOUR, J. Genetics 46, 180 (1945).

¹ E. C. GROB, W. H. SCHOPFER und G. G. PORETTI, Int. Z. Vitaminforsch. 23, 484 (1952). – E. C. GROB und R. BÜTLER, Exper. 10, 250 (1954).

² M. ARNAKI und Z. STARY, Biochem. Z. 323, 367 (1952).

