cal shifts of Auger lines are of much larger magnitude than that of core electron lines for a particular system[3]. It is, therefore, advantageous to measure the Auger chemical shift in cases where the ESCA chemical shift is not appreciable. The Ga metal-oxide system is one such example where the shift of Ga $2p_{3/2}$ core level from oxide w.r.t. the metal[3] is less than 1 eV. A drop of gallium metal exposed to air was spread on an Al-metal foil coated with adhesive. The ESCA spectrum in the BE range, 1110–1130 eV was obtained. There was only a single peak of Ga $2p_{3/2}$ with BE value that corresponds to gallium oxide. However, the asymmetry of the peak indicated that there might be another peak on the lower BE side of the observed peak. Next, we recorded the spectrum in the BE range, 180-200 eV, in order to obtain the most intense Auger line $L_3M_{4,5}M_{4,5}$ of gallium. The spectrum showed two peaks at energies 184.6 eV and 190.2 eV. Both are Auger lines pertaining to $L_3M_{4,5}M_{4,5}$ transition. The line at lower BE could be assigned to Ga-metal while that at higher BE to Ga_2O_3. The energy difference between these two peaks is seen to be 5.6 eV which is the Auger chemical shift of Ga_2O_3 w.r.t. Ga-metal. Therefore it is advantageous to determine the Auger chemical shift for systems that do not have adequate ESCA chemical shifts.

13 October 1981

1. Siegbahn, K., Nordling, C., Fahlman, A., Nordberg, R., Hamrin, K., Hedman, J., Johansson, G., Bergmark, T., Karlsson, S. E., Lindgren, I. and Lindberg, B., *Nova Acta Regiae Soc. Sci.*, Upsaliensis, 1967, Ser. IV, **20.**
2. Wagner, C. D. and Biloen, P., *Surf. Sci.*, 1973, **35**, 82.
3. Wagner, C. D., *Disc. Faraday Soc.*, 1975, **60**, 291.

MUTATION INDUCTION BY BENOMYL IN *SACCHAROMYCES CEREVISIAE*

P. Gunasekaran and P. Tauro*
Department of Microbiology, School of Biological Sciences, Madurai Kamaraj University, Madurai 625 021, India.

* Department of Microbiology, Haryana Agricultural University, Hissar 125 004, India.

Benomyl (methyl 1, butyl carbamyl benzimadazole 2-carbamate) is a systemic fungicide with a wide range of antifungal activity. There are reports of this chemical as a mutagen in *Escherichia coli, Salmonella typhimurium* and *Fusarium oxysporum* and induces gene conversion in yeast and in *Neurospora crassa*[1-4]. In this communication, we report that benomyl is a strong mutagen in yeast *Saccharomyces cerevisiae*, only under growing conditions and does not cause inactivation or mutation in resting cells.

S. cerevisiae 2180-1 A a g^+ was from the yeast stock centre, University of California, Berkeley, California and was maintained on YEPD agar slants (yeast extract, 0.5%, peptone 1%, dextrose 2% and agar 2%). Benomyl (Benlate, 99% WP) was a gift from M/s DuPont, USA. To test if benomyl is mutagenic in yeast, cells were suspended in either synthetic growth medium (yeast nitrogen base 0.67% and glucose 2%) or in phosphate buffer (pH 7.0, 0.1 M) containing 10 μg/ml of benomyl and incubated at 30° C on a rotary shaker. At intervals 0.1 ml of the sample was withdrawn and diluted with distilled sterile water and plated on YEPD agar plates and incubated at 30° C for 72 hr. After determining the number of survivors, the plates were overlayered with 1% agar containing 0.1% tetrazolium hydrochloride to detect the number of petite mutants[5].

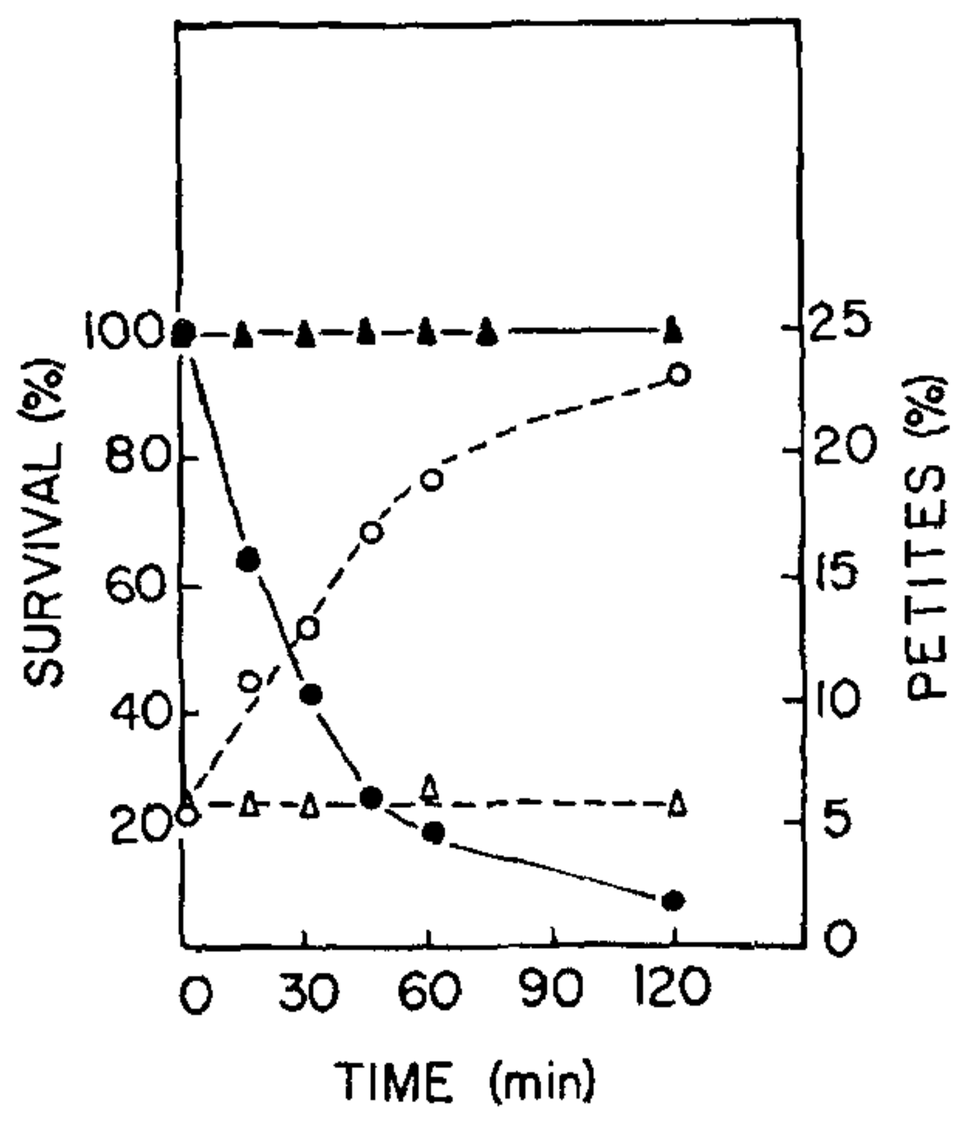

Figure 1. Effect of benomyl on induction of petite mutation. △, Percentage of survival petites in phosphate buffer (pH 7.0, 0.1 M). ○, Percentage of survival petites in synthetic growth medium. ▲ Percentage of survival in phosphate buffer (pH 7.0, 0.1 M). ● Percentage of survival synthetic growth medium. The concentration of benomyl was 10 μg/ml.

The number of survivors when treated with benomyl either in phosphate buffer or in growth medium (for 120 min) is shown in figure 1. Benomyl does not inactivate the resting yeast cells but strongly inactivates cells suspended in the growth medium. However, no growth occurs during the 120 min of

exposure. It, therefore, suggests that active cell metabolism is necessary for benomyl to be an inactivating agent. Benomyl also induces mutations only when present in the growth medium and not in resting cells. As seen in figure 1, the number of petites remains constant when exposed to benomyl in phosphate buffer but the number increases to a maximum of 23.5% when treated in growth medium. Mutation induction and inactivation is neither due to the pH of the medium (5.5) nor due to the presence of glucose since cells suspended in phosphate buffer (pH 5.5, 0.1M) and containing glucose (2%) show no inactivation.

TABLE 1

Mass complementation analysis of benomyl induced petite mutants with 9B ϱ^-

	Total number of mutants tested	Complementation with 9B ρ^-	
		+	−
Control	108	0	108
Benomyl induced	132	31	101

To determine whether benomyl induces both segregational and cytoplasmic respiratory deficient mutations, a larger number of randomly selected mutants were subjected to mass complementation test against a standard cytoplasmic petite 9B, ϱ^- (table 1). Out of 108 petites isolated from cells suspended in phosphate buffer, none complemented with the tester while out of 132 benomyl induced mutants, 31 complemented with the tester strain (22.7%). It therefore appears that about ¼ of the respiratory deficient mutations induced by benomyl are of segregational type.

Benomyl like in *Ustilago maydis* induces cell inactivation only in growth medium[6]. Being a purine analogue it is likely that the cell inactivation occurs only as a consequence of incorporation of this analogue during the DNA replication. It appears to be a potent mutagen for the induction of segregational respiratory deficient mutations.

The financial assistance received by one of us (PG) from the Council of Scientific and Industrial Research is gratefully acknowledged.

9 December 1981

1. Dassenoy, B. and Mayer, D. A., *Mutation Res.*, 1973, **21**, 119.
2. Kappas, A. G., Green, M. H. L., Bridges, B. A., Logers, A. M. and Muriel, W. J., *Mutation Res.*, 1976, **40**, 379.
3. Sieler, S. P., *Mutation Res.*, 1975, **32**, 157.
4. Siebert, D., Zimmerman, E. K. and Lamperele, E., *Mutation Res.*, 1970, **10**, 533.
5. Ogur, M. R., Jhon, S. and Jagai, S., *Science*, 1957, **125**, 928.
6. Clemons, G. P. and Sisler, H. D., *Pestic. Biochem. Physiol.*, 1971, **1**, 32.

ANTI-INFLAMMATORY ACTIVITY OF LAWSONIA INERMIS

SUJATA SINGH, N. M. SHRIVASTAVA, N. T. MODI, A. Q. SAIFI
Department of Pharmacology, Gandhi Medical College, Bhopal 462 001, India.

NADKARNI[1] has reported the use of Lawsonia inermis as an anti-inflammatory agent. Keeping in view the anti-inflammatory action of the leaves, an attempt has been made to evaluate this activity by standard techniques.

Powdered shade dried leaves of the plant (100 g) were extracted with 250 ml of 95% ethyl alcohol for 24 hr in a soxhlet. It was cooled, filtered, evaporated on water bath under reduced pressure and the residue obtained. The extract was tested for alkaloid, glycoside and saponin as per standard tests. A positive test was obtained for glycoside as the hydrolysed products of the extract decolourised Benedict's solution. Further study of isolation of glycoside is in progress.

The aqueous suspension of the extract of 10 mg/ml was prepared and was used as a stock solution. The pH of the suspension was adjusted to 7.0 with 10% sodium hydroxide.

The anti-inflammatory activity of alcoholic extract was tested in albino rats by Brodie's hind paw oedema test[2], Granuloma pouch test[3] and Lint pellet test[4,5]. The alcoholic extract of the drug was given by *intraperitonial* route.

Inflammation was produced by injecting 1% carrageenin solution in a dose of 0.2 ml each in the plantar aponeurosis of albino rats for Brodie's hind paw oedema test. The granuloma pouch test was performed by producing granuloma pouches by injecting 0.5 ml of 1% croton oil dissolved in almond oil. Lint (cotton) pellet test was performed by inserting lint cloth pellets (weighing approximately 2 mg) in the axillary area. The degree of deposition of granulation tissue on lint pellet indicated the degree of inflammation. The observations are shown in table 1.