

turbidity in the tubes containing lower concentrations of penicillin particularly when in the presence of nucleic acid alone there is as much depth of turbidity due to growth as is indicated by four *plus* signs. There is thus a clear contradiction between the observation and explanation which Ganapathi, *et al.*, have described. Thirdly the statement in the last paragraph of the note Ganapathi *et al.* imply that penicillin cannot be destroyed at pH 7.0 under the conditions they describe. It is now well known that as the pH tilts to alkalinity penicillin gets progressively destroyed.

It is also well known that penicillin acts best on young actively growing cells. Since the optimum growth of pathogenic organisms happens to be 37° C., penicillin is more active at 37° C. than at lower temperatures on bacteria. In the case of insensitive bacteria like *Esch. coli* requiring high minimum inhibiting concentrations of penicillin, even a very high concentrations of nucleic acid 1/100 cannot reverse the inhibition brought about by penicillin, while this is actually achieved by the magnesium salt of nucleic acid.⁴ We have indeed found that the organisms which had become nonviable by contact with bacteriostatic concentration of penicillin were actually rendered viable by added nucleic acid¹ a fact which clearly indicates the lack of any correlation between pH effects and capacity of nucleic acid to antagonise penicillin bacteriostasis. It may thus be seen that under the conditions used, pH effects and temperature effects do not orientate the influence of nucleic acid on penicillin bacteriostasis and our conclusion that nucleic acid reverses the bacteriostatic effect of penicillin on *Staph. aureus* seems quite warranted. It is interesting to note in this connection that Pratt and Dufrenoy⁵ have offered a very sound and convincing explanation of our observations.

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1. Pandalai, K. M. and George, M., *Brit. Med. Jour.*, 1947, **2**, 210; *Curr. Sci.*, 1947, **16**, 312.
2. Ganapathi, K., Sadasivan, V., Barucha, F. D., and Radhakrishnan, M. R., *Curr. Sci.*, 1948, **17**, 263.
3. Benedict, R. G., Schmidt, W. H., Coghill, R. D., and Oleson, A. P., *Jour. Bact.*, 1945, **49**, 85.
4. George, M., and Pandalai, K. M., *Brit. Med. Jour.*, 1948, **1**, 1028.
5. Pratt, R., and Dufrenoy, J., *Bact. Reviews.*, 1941, **12**, 93.

We have carefully considered the note of Pandalai and George and have to report that our original contention stands. In the note of Pandalai and George, it is claimed that "they have shown that nucleic acids possess the property of antagonising penicillin action" and they have "suggested that penicillin interferes with certain phases in the metabolism of the organisms where nucleic acids feature either as metabolites helping cell division or function as respiratory catalysts or both." If this sweeping assertion be true, then nucleic acids should antagonise the action of penicillin *in*

vitro in a very significant way. We have shown that this is not the case. Our experiments have been misinterpreted by Pandalai and George. When the broth contains penicillin in concentrations ranging from 0.05 units to 16 units per ml. *along with* nucleic acid or its sodium salt in concentrations ranging from 1/10000 to 1/200, we could observe no reversal of bacteriostatic action against *Staphylococcus aureus*. When nucleic as the free acid is used, in the borderline concentration of 0.05 units per ml., there is an apparent slight reversal of penicillin bacteriostasis, which we have suggested to be due to the destruction of penicillin and consequent lowering of the concentration to an extent at which it is not bacteriostatic. Pandalai and George now question this destructive action of nucleic acid. We have experimental evidence to support our conclusion. To the broth was added in one case penicillin *plus* sodium salt of nucleic acid in the other penicillin *plus* free nucleic acid. These were incubated at 37° C. overnight and the penicillin concentration in the two sets of tubes estimated by the iodine titration method. In the broth containing sodium salt of nucleic acid about 90% of the added penicillin could be recovered, while in those containing free nucleic acid only 40 to 50% of the added penicillin could be recovered. Thus, when in the borderline concentration of 0.05 units per ml. about 50% of the penicillin is destroyed, the remaining quantity is not sufficient to show the full degree of bacteriostasis. Where higher concentrations of penicillin are used, even after the destruction of 50% of the added penicillin, there is still enough penicillin left over in the broth to show bacteriostasis. There is no contradiction in the result, as has been suggested by Pandalai and George. All the results reported by Pandalai and George are due to this destruction of penicillin by nucleic acid and no experimental evidence has been advanced to prove their elaborate theory.

Pandalai and George claim that cultures rendered nonviable by "prolonged contact with penicillin" are rendered viable by the addition of nucleic acid. According to their experiments, the cultures have been kept in contact with penicillin for 3 and 6 hours only. We have repeated the experiment and have found that under these conditions all the organisms are not rendered nonviable. In our experiments, one mm. loopful of a 24-hour old culture of *S. aureus* was inoculated into 5 ml. of standard broth containing different concentrations of penicillin. These were incubated for 3, 6 and 18 hrs. at 37° C and at the end of these periods, 0.1 c.c. of these cultures were transferred to 5 ml. of sterile broth and these tubes were incubated for 24 hours. Thus by reducing the concentration of penicillin, the viable organisms present are allowed to grow. We have found growth in all the tubes in which penicillin was used in concentrations of 0.1 to 0.3 units per ml. in the original tubes. Thus it is clear that even with 18 hours contact with penicillin, all the organisms do not become nonviable as claimed by Pandalai and George. So the organisms that are left over start multiplying when nucleic acid is added to the broth which destroys penicillin.

Thus Pandalai and George have not produced any proof to support their contention. They refer to the review of Pratt and Dufrenoy; these authors do not confirm the observations of Pandalai and George; they only comment on them. We primarily question the correctness of the results of Pandalai and George.

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SEX INHERITANCE IN *LUFFA ACUTANGULA*

As a result of single plant selection studies, some true breeding cultures for various characters have been established from the local material of *Luffa acutangula* and two of such selections which differed in their sex condition formed the basis of the present genetical studies, viz., *Jhingli No. 1* which is monoecious and *Satputia No. 1*, hermaphrodite.

Crossing was attempted both ways, but it succeeded with *Satputia No. 1* as female only. Sixteen F_1 hybrids were raised, all of which proved to be monoecious (producing male and female flowers on the same individuals). Very rarely hermaphrodite flowers were also observed on the same individuals. Pollen formation and seed setting were found to be quite normal in these hybrids.

F_2 generation was raised from selfed seeds of four different F_1 hybrids. In all 266 hybrids were raised and studied and they could be distinctly classified into three different classes (Table I), as explained below:

- (a) *Monoecious plants*: This class of hybrids showed the sexes in different flowers, male and female, on the same individuals. They exhibited variation in two directions, viz., (1) in the stage at which the female elements appeared, as in some of them the female flowers appeared soon after the male ones, whereas in others at a very late stage, and (2) in the appearance of hermaphrodite flowers in addition to male and female ones. In some of them such flowers appeared in greater number than in others.
- (b) *Female plants*: This class of hybrids produced female flowers only.
- (c) *Hermaphrodite plants*: This class of hybrids produced hermaphrodite flowers only.

TABLE I

Segregation in F_2 population for different sex forms in a varietal cross of *L. acutangula*

	Segregation in F_2 generation			Total No. of F_2 hybrids
	Monœcious	Female	Hermaphrodite	
Observed	203	51	12	266
Expected on 12:3:1	199.5	49.875	16.625	266

$$X^2 = 1.372 ; p > 0.50$$

The F_2 segregation indicates that there are possibly two independent factor pairs, res-

possible for sex inheritance in this cross, designated as AA BB: The factor pair AA controls the expression of sex elements, both male and female, distributed in separate flowers on the same plant (monoecious condition). Where this factor for monoecious condition is lacking (in plants which are aa), the plant would be hermaphrodite, but this expression is influenced by the other factor B which controls the expression of female sex only and in its absence the plant again becomes hermaphrodite. But the factor B is ineffective in the presence of A which may be said to be epistatic to B. Sex in plants which are aa is then exclusively determined by B. Thus in the absence of A the hybrids with the constitution aa BB or aa Bb would be female plants and those with Aa or AA would be monoecious whether B is present or absent. In the absence of both A and B, the plant with the constitution aa bb will be hermaphrodite (the double recessive genotype). The constitution of the parents will thus be aa bb in the case of *Satputia No. 1* and AA BB in the case of *Jhingli No. 1*.

In short, sex inheritance in this cross is controlled by two independent factor pairs, one of which determines the expression of both sexes, whereas the other one controls female sex only, the first one being epistatic in action. In the absence of both, an individual becomes hermaphrodite.

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PRODUCTION OF P-CYMENE FROM CARENE*

CARENE is a raw material for the production of p-Cymene (i).^{1,2,3,4} The work reported in this note is on the disproportionation of this hydrocarbon to (i) over Fe_2O_3 -gypsum catalyst.

The catalyst is prepared as follows: 17.5 gm. of $FeSO_4 \cdot 7H_2O$ is dissolved in 500 c.c. water; added to the solution a few c.c. con. nitric acid and gradually heated. To the fully oxidised solution added ammonium chloride and then dilute ammonia carefully with stirring, until the precipitate is permanent. The precipitate is washed by decantation several times with water. Mixed in 300 gm. of well-washed gypsum pieces, 8-10 mesh size. It is then dried on a water-bath with constant stirring, and finally ignited to the oxide stage in a fire-clay crucible.

Carene (b.p. 163-68° C./745 mm. d_{15}^{15} : 0.8468, n_D^{20} : 1.4716) fractionally distilled from Indian turpentine (*P. longifolia*), is passed over the catalyst bed, 60 cm. long, occupying a total space of 234.0 c.c.; time of contact is 3 hours for 100 c.c. carene.

The pyrogenic unit is described in a previous communication.⁴

Identification of (i) is by oxidation by chromic acid to terephthalic acid (dimethyl ester, m.p. 140° C.).

Table I gives the analysis of the major fraction containing (i) after one fractionation; Table II gives the result of the fractional