

concurrent wastage by such attack as shown by Nozaki and Bartlett.³

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1. Goldberg, Hohensten and Mark, *J. Poly. Sci.*, 1947, 2, 503. 2. Price, *Reactions at Carbon-Carbon Double Bond*, p. 103, Interscience Publishers, New York, 1946. 3. Nozaki and Bartlett, *J. Amer. Chem. Soc.*, 1946, 68, 1686.

PYROGENIC DECOMPOSITION OF CARENE IN THE PRESENCE OF COPPER AND ALUMINIUM CATALYSTS

ON passing the vapours of carene (b.p. 163-68° C./745 mm., d_{15}^{20} : 0.8468, n_D^{20} : 1.4716, from Indian turpentine, *P. longifolia*) through copper turnings heated to 100° ± 15° C.¹ in the pyrogenic unit previously described,² at an hourly liquid space velocity: 0.14, the terpene hydrocarbon was decomposed. Among the reaction products were 8.8% gases and 89.2% oil. 20.3% of the pyrolysate distilled between 173-78° C./745 mm. (d_{15}^{20} : 0.8697, n_D^{20} : 1.4775) and contained p-cymene.

With aluminium turnings, the gases amounted to 12.3% and oil 83.7%. The yield of the 173-78° C./745 mm. fraction was reduced to 18.5% (d_{15}^{20} : 0.8699, n_D^{20} : 1.4797).

The experiments suggest that a furnace of copper³ or aluminium will have a gentle accelerating effect on the disproportionation of carene to p-cymene.

Tech. Chem. Lab., JAMES VERGHESE.
Forman Christian Coll., H. K. SONDHI.
Lahore, BHARAT BHUSHAN.
December 21, 1948. M. L. JOSHI.

1. See Orlov, "Pyrogenic oxidation of turpentine in the presence of a copper catalyst," *Ukrainski Khem. Zhurnal*, 1926, 2, 1; *Chem. Zentr.*, 1926, II, 660. 2. Sondhi, Bhushan, Gulati and Joshi, *J. Indian Chem. Soc. (Ind. and News Ed.)*, 1947, 10, Nos. 1 & 2, 17. 3. See Kirkpatrick (to Hercules Powder Co.), U.S. Pat. June 25, 1946, 2, 402, 898.

ON THE NATURE OF INHIBITION OF ERYTHROCYTE PYROPHOSPHATASE BY VERONAL-ACETATE BUFFER

It was previously reported¹ that the erythrocyte pyrophosphatase is greatly inactivated by incubation for ½ hr. with M/35 veronal-acetate buffer alone, prior to the addition of the substrate and the activator; and that the presence of the activator (Mg^{++} ion) protect the enzyme from such inactivation to a certain extent. On further study on the nature of the inactivation it was discovered that both the buffer constituents, viz., veronal and acetate, are themselves responsible for the inactivation to a great extent, the inactivation due to heat (38° C.) being comparatively small (Table).

1 ml. of 1 in 20 haemolysate (human erythrocytes) was incubated with 3 ml. of the inhibitor of different concentrations for varying periods of time, and then the enzyme activity was determined by adding 1 ml. of 0.1 M $MgCl_2$ and 0.5 ml. of 0.01 M sodium pyrophosphate. Period of hydrolysis 15 mins. pH -7. Temp. -38° C. Percentage of inhibitions were calculated from the orthophosphate content of the trichloroacetic acid filtrates.

TABLE

Enzyme incubated with	Per cent. inhibition produced		
	preliminary incubation period		
	15 mins.	30 mins.	60 mins.
0.2 M Sodium acetate	60	80	..
0.1 M " "	..	68	87
0.04 M " "	..	33	62
0.04 M Sodium veronal	..	76	88
0.02 M " "	..	42	62
0.01 M " "	..	22	34
Water	4	6	12

It was further observed that the pyrophosphate ion affords better protection of the enzyme than the Mg ion against the inhibition due to the buffer constituents.

A number of substances related to the buffer constituents were studied and varying degrees of inhibition were observed. All the solutions were adjusted to pH 7, and after incubating 1 ml. of the enzyme with 3 ml. of the inhibitor of varying concentrations for different time periods, the