SPECIAL ARTICLES

ALCOHOLIC FERMENTATION BY FUSARIA JUICE OBTAINED WITH A WET CRUSHING MILL

THE study of certain aspects of the mode of action of cell-free yeast juices from the point of view of colloid chemistry was introduced several years ago,[1,2,3] and led, among other developments, to the experimentally established rule that enzymes in juices and within the cell may change their activity in accordance with the degree of dispersion of the accompanying carriers. Miss M. G. Macfarlane[4] presents an impressive, though presumably far from complete list of differences in behavior between the living yeast cell and the so-called cell-free extracts in regard to the mechanism of alcoholic fermentation. The fermenting cell-free juices generally used (prepared according to Buchner, Lebedew or Lipmann) necessarily exhibit partial or complete suppression of activities or losses of a single or several members of the enzyme systems involved. This accounts for the well-known fact that the harmonious order of the living cell and the phase sequence of enzyme action within it become disorganized due, *e.g.*, to dilution, disruption of carrier systems because of possible denaturation, proteolysis, etc.

A cell system which exhibits, beyond doubt, a measurable phosphorylation and subsequent dephosphorylation of carbohydrates during fermentation is that of the Fusaria. It was deemed justified, therefore, to attempt to investigate an artificial enzyme system from *Fusarium lini Bolley* extracted with the aid of the Booth-Green wet crushing mill.[5] This juice, due to the manner of its preparation, could, if found to be at all active, be compared to a large extent with the juice obtained long ago by Dixon and Atkins,[6] but scarcely investigated by them.

After applying a slight mechanical improvement to the mill, we obtained a juice which can be described as follows. It is opaque; its color varies from light yellow to reddish-brown, according to the distribution of the pigment present.[7] Its pH, prepared with water, is in the neighborhood of 5.7 (the internal cell pH of *Fusarium lini Bolley* lies between 6.0 and 6.1[8]); its relative viscosity measured at 25.3° is 1.168, the determination on the juice having been made a few days after preservation at 6°.

The measurable esterifying capacity of the juice at 28°, as determined by the method of Fiske and Subbarow,[9] amounts under our experimental conditions to about 35 to 40 per cent. of the phosphorylation in the living cell system. In some cases this appears to have been preceded by a dephosphorylation of the organic phosphate donators originally present in the living Fusaria cells.[10] The esterification brought about by the quantity of inorganic phosphate present amounted to about 3 to 4 per cent. The quantity of CO_2 obtained by the action of the zymases compared favorably with that of the living system.

Judging from the data available at present, and taking into consideration the fact that the crushing of the cell makes uniform the highly different pH values of closely organized cell particles,[11] the cell-free Fusaria juice obtained with the wet crushing mill seems to have furnished us so far with an enzyme system which does not exhibit deficiencies or distortions of kind or magnitude as compared with the usual maceration juices obtained from yeasts.

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THE ACTION OF TYPE-SPECIFIC ANTIBODY UPON THE PULMONARY LESION OF EXPERIMENTAL PNEUMOCOCCAL PNEUMONIA[1]

ALTHOUGH the effectiveness of type-specific antiserum in the treatment of pneumococcal pneumonia is well established, the action of antibody upon the pulmonary lesion is not clearly understood. Considerable evidence[2,3,4] has been advanced supporting the view that antibody can not penetrate areas of consolidation within the lung. Recently Kempf and Nungester[5] have studied the penetration of antipneumococcal immune bodies into pneumonic lesions produced experimentally in rats. Using both horse and rabbit antiserum, they were unable to demonstrate the presence of antibody in the lungs following intravenous treatment. They concluded that, even were it found to penetrate the consolidated area, the antibody could not be expected to accumulate in sufficient concentration to neutralize the pneumococcal polysaccharide present in the alveoli.

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