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AN EXAMINATION OF BARK OF *QUERCUS SEMICARPIFOLIA*

Quercus semicarpifolia, an important species of Oak belongs to Fagaceae family[1]. Crude extract of bark of this plant has been reported active against various diseases, *viz.*, asthma, diarrhoea, etc., and is given as diuretic in gonorrhoea and as astringent in indigestion[2]. The bark was subjected to chemical analysis as follows.

Well dried and finely powdered bark (5 kg) of *Q. semicarpifolia* was soxhleted with petroleum ether (15 l) for 48 hr and the extract was concentrated under reduced pressure and was passed through a glass column packed with silica gel G. It was then eluted with benzene. Three fractions which exhibited single spot on tlc plates were further purified over preparative tlc plates and identified as Fridelin, -sitosterol and Taraxerol on the basis of their m.p., m.m.p., co-tlc and superimposable I.R. and mass spectra. This is the first report on the presence of these three constituents in the bark of *Q. semicarpifolia*.

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A PRELIMINARY STUDY ON THE ROLE OF PYRIDOXINE IN LIPID METABOLISM IN *ASPERGILLUS NIDULANS*

THE role of pyridoxine in variuos metabolic processes in animals has been studied in detail by many workers[1-6]. Deficiency of pyriodoxine has been observed to affect cellular growth (production of lymphopaenia[4]) and inhibit viral replication[5] involving possibly nucleic acid metabolism. The effect of pyridoxine deficiency in phospholipids[6] and in the changes of galacto lipid fatty acids in the brain[7] in the case of rats have been recently reported. In view of the fact that earlier studies have been made on the variation of lipid metabolism in pyridoxine deficient animals it was considered interesting to examine the same in cells which have lost the ability to synthesise pyridoxine (*i.e.*, pyridoxineless mutants) and the results of a preliminary study are presented here.

The wild strain (Y) and pyridoxineless mutant with yellow conidia (y_1 : *pyro*$_8$) of *Aspergillus nidulans* have been used for the investigation. While the normal (wild) strain was cultured in liquid minimal/agar medium[8], the mutant was cultured in the same containing 4 μg of pyridoxine per 50 ml of the medium for seven days. The mycelia were then harvested, filtered, washed well with distilled water and once with 0·1 M Tris-HCl buffer (pH 7·0), dried between folds of filter paper and kept at 0°C for use within 24 hr. The amount of glycogen[10], protein[1], DNA[11], RNA[12] and total lipids[13] were estimated adopting standard procedures. The lipids were further separated by column (DEAE cellulose) chromatography[14] and the fractionated components (sterols[15], triglycerides[16], fatty acids[15] and phospholipids[18]) were estimated. They were also separated by subjecting them to Thin Layer Chromatography[19] on silica gel and the individual components identified wherever possible by comparison with known compounds. Besides sterol esters, mono and diglycerides and their alkyl derivatives, triglycerides and free sterols, a number of lipid components not identified earlier could be detected whose characterisation could not be done due to paucity of material.

It may be seen from Table I that there is definite difference in the lipid components of the wild and pyridoxineless mutant showing the influence of pyridoxine in the lipid metabolism of these cells. The mycelial weight, DNA as well as RNA content in the mutant is less than in the wild; a similar decrease in nucleic acids has been reported[20] in pyridoxine deficient rats. It is also observed that the cellular lipid level (especially the triglycerides and the unidentified lipid components) is increased in the mutants indicating the role of pyridoxine in their metabolism. However, further work on a larger scale is needed to identify the nature of all the lipid components and