solid (20 g.). This was boiled with methanol, filtered, and recrystallised from glacial acetic acid yielding a crystalline colourless solid, T.L.C. pure, m.p. 297–98°. It did not give any colour with alcoholic ferric chloride or Mg and HCl but when it was treated with sodium amalgam in ethanol, left overnight and acidified, a deep pink colour developed showing its isoflavone nature. It gave a deep blue colour when heated with gallic acid and concentrated sulphuric acid at 80° showing the presence of methylene-dioxy group ; u.v. spectrum $\lambda_{max.}^{MeOH}$ 256 mμ (log ε 4·67), 293 mμ (log ε 4·06). The above physical as well as chemical study revealed that the compound is ψ-baptigenin (7-hydroxy 3', 4' methylene-dioxy isoflavone). The identity was confirmed by the preparation of its methyl ether and acetate.

Leaves.—Air-dried leaves (800 g.) were similarly extracted with the above-mentioned series of solvents. The light petroleum extract gave only wax and chlorophyll. The acetone and alcoholic extracts gave ψ-baptigenin in quite high yields (2%). After removing it, the mother liquors were treated with neutral and basic lead acetates ; lead salts yielded quercetin and kæmpferol in low yields.

Since ψ-baptigenin was present in considerable amounts in the leaves of *D. lanceolaria* it was considered desirable to test whether it could be the main compound responsible for the beneficial properties of the leaves in arthritic ailments. A large sample was submitted to the Post-Graduate Institute of Indian Medicine and the report of tests[2] on animals and clinical trials on human patients confirm its potency.

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EFFECT OF ADRENERGIC BETA RECEPTOR BLOCKING DRUGS ON RAT BRAIN 5-HT LEVEL

Adrenergic β-receptor blocking drugs possess important effects on the central nervous system. Propranolol has been reported to possess central depressant and muscle relaxant as well as antitremor actions.[6,7,2] N-isopropyl-β (4-methanesulphonamidophenyl) ethanolamine (MJ 1999) also possesses a depressant action on the brain.[5] A recent adrenergic β-receptor blocking agent d-n-isopropyl-p-nitrophenyl ethanolamine (d-INPEA), however, has central excitatory effect.[6] Propranolol[5] and d-INPEA (unpublished observation) inhibit monoamine oxidase (MAO) activity, *in vitro*. Since this enzyme is concerned with the destruction of 5-hydroxy-tryptamine (5-HT), an amine which might be concerned with the functional activity of the brain, the present work was undertaken to study the effect of adrenergic β-receptor blocking drugs on brain 5-HT level in albino rats.

Propranolol, MJ 1999, d-INPEA or normal saline were injected intraperitoneally into albino rats (150–200 gm.). The animals were decapitated 1 hour later, after the behavioural effects of the drugs manifested, and their brains put in ice-cold acetone. 5-HT was extracted by the method of Amin, Crawford and Gaddum (1954)[3] and assayed on the rat stomach fundus by the method of Vane (1957).[8]

The effects of drug treatment on brain 5-HT level has been shown in Table I.

Table I
Effect of adrenergic β-receptor blocking drugs on rat brain 5-HT level

Drugs	Dose mg./kg.	No. of experiments	Brain 5-HT content μg./gm.	p value
Control ..	..	10	0·40	..
Propranolol	10	6	1·05	<0·05
MJ 1999 ..	80	6	0·92	<0·1
d-INPEA ..	80	6	0·45	>0·9

From Table I it may be seen that propranolol and MJ 1999 cause a rise in brain 5-HT. These drugs have also been reported to depress the central nervous system. However, only propranolol has been shown to possess MAO inhibiting action. It therefore appears that the rise in brain 5-HT may be due to the non-specific depressant action. Such an effect has also been reported after other sedatives like barbiturates, meprobamate and morphine.[1] The greater increase in 5-HT after propranolol may be due to its additional enzyme inhibiting effect. d-INPEA, in spite of its inhibiting action on MAO *in vitro*, does not elevate brain 5-HT which could be due to lack of enzyme inhibition, *in vivo*.

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PECTIN *TRANS*-ELIMINASE ACTIVITY IN *CYTOPHAGA*

ALBERSHEIM, Neukom and Deuel[1] discovered the enzyme *trans*-eliminase in the commercial pectinase "Pectasin R-10". This enzyme splits α, 1-4 glycosidic bonds in pectin by a *trans*-elimination reaction and the product of its degradation has a characteristic absorption maximum at 230–235 mμ. Widespread distribution of this enzyme in bacteria, actinomycetes and fungi has been reported. Also, according to the substrate specificity exhibited, more than one enzyme have been recognised and these have been now designated as either pectin *trans*-eliminase[2] or polygalacturonic acid *trans*-eliminase.[3]

The presence of *trans*-eliminase activity in *Corynebacterium barkeri*, *Flavobacterium* sp., *Micrococuss* sp. and *Arthobacter* sp., has previously been reported from this laboratory. It was shown that several species of *Streptomyces* also exhibit pectin *trans*-eliminase activity. Interestingly, the enzyme was found to be produced even by protozoa. Recently, Agate, Jayasankar and Bhat[4] have traced the literature and discussed pectin *trans*-eliminase in detail in their review in this journal.

The present note deals with the detection of pectin *trans*-eliminase and polygalacturonic acid *trans*-eliminase in some *Cytophaga* species isolated from soil and water samples. Needless to state that pectinolytic properties have not been attributed to the species of this genus. Surprisingly, the culture filtrates of *Cytophaga deprimata*, C. *albogilva* and C. *johnsonii* failed to show pectin methyl esterase and polygalacturonase activity when tested by $CaCl_2$-gel formation and alcohol precipitation respectively; however, the culture filtrates revealed pectinolytic activity when tested by the cup-plate method described by Nagel and Vaughn.[5] The enzymes present in these species were characterised to be pectin *trans*-eliminase and polygalacturonic acid *trans*-eliminase when tested by a method similar to that described by Nagel and Vaughn[6] which consisted of adding metabolic filtrate to a buffer substrate (pH 8·0) containing 0·001 M calcium chloride + pectin or polygalacturonic acid as substrates and examination of the reaction mixtures (after incubation for 24 hours at room temperature—25-27° C.) for their absorption spectrum in the range of 220–250 mμ. The reaction mixtures showed an absorption peak at 235 mμ. Confirmation of the finding was sought and indeed derived from the thiobarbiturate reaction of the break-down product, *viz.*, digalacturonide which gave an absorption maximum at 548 mμ.[7]

The extent of decomposition of pectin by *Cytophaga* species was then determined by measuring the residual pectin in culture filtrates according to the method of Kaiser.[8] The decomposition ranged from 10 to 40% within 15 days (stationary cultures). Further work is in progress and, in the meantime, it is hoped that this first report on the *trans*-eliminase in the *Cytophaga* species would be of interest to the investigators on these bacteria as well as these enzymes.

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NOTE ON THE OCCURRENCE OF *OSTREA TALPUR* VRED., IN THE INTER-TRAPPEAN BEDS AT DUDDUKURU, NEAR RAJAHMUNDRY, A.P.*

THE note records the occurrence of *Ostrea talpur* Vred., a characteristic Lower Ranikot oyster from Sind (W. Pakistan) in the Inter-trappean Beds exposed about 1·5 Km. South-East of Duddukuru (17° 02′ 15″ : 81° 35′ 45″; 65 G./12), near Rajahmundry, Andhra Pradesh. The fossils occur in a 10 m. thick band of buff