acetazolamide and were both decreased by ethosuximide (an effect previously observed with phenobarbitone). The thiol-group inhibitors iodoacetamide (100 μ M) and 5,5'-dithiobis-(2-nitrobenzoic acid) (100 μ M) did not alter the rate of xylose uptake over a 9 min incubation period. However, both inhibitors abolished the stimulatory effects of acetazolamide and ethosuximide.

The significance of these results will be discussed.

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Microbial Metabolism of Alkylbenzenesulphonates

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Alkylbenzenesulphonates are a group of detergent compounds in common domestic and industrial usage. Several reports (Nelson, McKinney, McAteer & Konecky, 1961; Huddleston & Allred, 1963; Swisher, 1963) have concluded that this group of substituted aromatic compounds is biodegraded, but previous research has almost exclusively been limited to whole-cell studies with a mixed microflora degrading, in many instances, an isomeric mixture of alkylbenzenesulphonates. This communication reports the enzymology of (1-phenylundecyl)benzene-*p*-sulphonate degradation by a pure culture of a *Bacillus* sp. elected for growth on alkylbenzenesulphonates as the major source of carbon and sulphur.

Continuous sampling experiments, after inoculation of the pure strain of *Bacillus* species into alkylbenzenesulphonate medium, indicated that the sequence of biochemical events was: (1) removal of the sulphonate grouping, probably at the cell surface, leaving free sulphite and a *p*-hydroxyphenylalkanoic acid in the medium; (2) assimilation of sulphite and its subsequent intracellular oxidation to sulphate plus reduction to sulphide; (3) assimilation of the *p*-hydroxyphenylalkanoic acid, which was subsequently degraded intracellularly by classical β -oxidation of the alkyl side chain followed by 'ortho' cleavage of the aromatic nucleus (Ornston & Stanier, 1964).

The specific activity of the desulphonating enzyme, which was assayed by following sulphite production (Robinson, 1965), was increased 100-fold by growth on (1-phenylundecyl)benzene-*p*-sulphonate over that observed in succinate-grown cells. The enzyme was repressed but not inhibited

by sulphite in the medium. Substrate-specificity studies implicated isosteric activation of the enzyme. Sulphite-cytochrome *c* oxidoreductase activities were also elevated sixfold after growth on alkylbenzenesulphonates, but adenosine phosphosulphate-cytochrome *c* reductase was constitutive in this *Bacillus*.

Enzymes associated with β -oxidation were recorded in alkylbenzenesulphonate-grown cells. The acyl-CoA acyltransferase present was active with a range of detergent isomers and β -hydroxyacyl-CoA dehydrogenase was also detected. The activity of isocitrate lyase, an enzyme characteristically induced in metabolic sequences producing acetyl-CoA, was 90-fold higher in alkylbenzenesulphonate-grown than in succinate-grown cells.

The aromatic nucleus produced by biodegradation of alkylbenzenesulphonate homologues containing an even number of carbon atoms in the side chain was identified by g.l.c. and t.l.c. as *p*-hydroxyphenylacetate. The equivalent aromatic nucleus from homologues with an odd number of carbon atoms in the side chain was identified as *p*-hydroxybenzoate. All the enzymes to degrade the appropriate aromatic by a classical 'ortho' pathway to succinate and acetate were induced to high activities in *Bacillus* after growth on the appropriate alkylbenzenesulphonate isomer.

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Metabolism of Aromatic Compounds by Fungi: Conversion of β -Carboxymuconolactone into 3-Oxadipate in *Aspergillus niger*

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The conversion of protocatechuate into 3-oxadipate was first shown to proceed via β -carboxymuconolactone [3-carboxy-4-hydroxyhex-2-enedioic acid (1 $\rightarrow$ 4)-lactone] in *Neurospora crassa* (Gross, Gafford & Tatum, 1956) and later incriminated as the lactone intermediate characteristic of fungal aromatic metabolism by Cain, Bilton & Darrah (1968). Prokaryotic organisms neither form β -carboxymuconolactone nor do they