protein-deficient purified diet (3% casein), phenobarbitone increases cytochrome *P*-450 only from 6 to 30nmol/g. of liver. This effect is only due to dietary protein, however, and re-feeding experiments indicate that the control of induction in protein-deficient animals may be altered so as to divert available protein away from less essential functions, of which the synthesis of cytochrome *P*-450 seems to be one.

The permissive effect of the factor in diet 41B has not been described before; its nature and possible mode of action will be discussed.

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Gut Bacteria and the Metabolism of Cyclamate in the Rat

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It has been established that cyclohexylamine is a minor metabolite of cyclamate (cyclohexylsulphamate) in the urine in man and animals (see, e.g., Golberg, Parekh, Patti & Soike, 1969; Oser, Carson, Nogin & Sonder, 1968).

We have found that Wistar albino rats kept on a normal diet and water excreted very little cyclohexylamine after a single oral dose of cyclamate, but rats kept for 3 months on the same diet but with water containing 0.5% calcium cyclamate (about 550mg./kg./day) developed the ability to convert cyclamate into cyclohexylamine. The latter rats (22) were given a single oral dose of calcium cyclamate (200mg.) and the urinary output of cyclohexylamine was determined by g.l.c. The urinary excretion of cyclohexylamine in 24hr. ranged from 0.7 to 35% of the dose (seven rats excreted more than 10% and 15 less than 10%). The cyclamate-treated rats (three) were given a single oral dose of [¹⁴C]cyclamate (50mg./kg.) and they excreted 93 (88–97)% of the ¹⁴C in 3 days, 64% in the urine and 27% in the faeces. In the first 24hr. after dosing an average of 58% of the ¹⁴C was excreted in the urine, 33% as unchanged cyclamate, 22% as cyclohexylamine and 3% as probably conjugated cyclohexylhydroxylamine. The faeces contained cyclamate and cyclohexylamine.

Three cyclamate-pretreated rats were given simultaneously [¹⁴C]cyclamate (10mg./kg.) intra-

peritoneally and unlabelled cyclamate (320mg./kg.) orally. The 24hr. urine was examined for cyclohexylamine by g.l.c. and scintillation counting. The conversion of [¹⁴C]cyclamate (given intraperitoneally) into cyclohexylamine was 0.9%, whereas that of unlabelled cyclamate (given orally) was 38%. About 90% of injected cyclamate was excreted in the urine in 24hr. compared with 58% when given orally.

When [¹⁴C]cyclamate was incubated for 1.5hr. with preparations of liver, kidney, spleen or blood from cyclamate-pretreated rats the formation of cyclohexylamine was no different from that of controls, i.e. less than 0.05%. The contents of the hindgut of the rats, however, converted 0.8–3.5% into cyclohexylamine under anaerobic conditions and 0.4–2.5% under aerobic conditions.

When the pretreated rats were taken off the cyclamate-containing diet, the ability to produce cyclohexylamine from cyclamate fell to a low level in 5 days.

These experiments suggest that the conversion of cyclamate into cyclohexylamine in the rat is carried out by the gut flora, which acquire this ability when the diet contains cyclamate. This ability is largely lost on removing cyclamate from the diet.

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The Physical Properties and Mechanism of Action of Deoxyxoglucarate Dehydratase from *Pseudomonas acidovorans*

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Deoxyxoglucarate dehydratase catalyses the conversion of D-4-deoxy-5-oxoglucarate into 2,5-dioxovalerate (2-oxoglutarate semialdehyde), CO₂ and water (Jeffcoat, Hassall & Dagley, 1968).

We report here the following physical properties of the enzyme from *Pseudomonas acidovorans* N.C.I.B. 10013 grown on glucarate: *S*_{20,w}⁰, 7.0s; *D*_{20,w}, 5.82 × 10⁻⁷ cm.²/sec.; $\bar{v}$, from the amino acid composition, 0.74 cm.³/g.; *E*_{cm}^{1%} at 280nm., 8.3; mol.wt. by sedimentation equilibrium, 118000; *S*_{20,w}⁰ in 6M-urea, 2.3s; *S*_{20,w}⁰ in 6M-guanidine