glucose was the carbon source. Parallel results were obtained in a study of the influence of α-methyl-glucoside upon the adaptation of *E. coli* to the oxidative metabolism of lactose and maltose. The glucoside would not itself support the growth of *E. coli*, nor was it metabolised, as measured by growth, oxygen consumption or the release of free glucose.

The adaptive formation, by stock cell suspensions[2], of the nitratase enzyme system (followed as previously described[3] except that the concentration of H-donor was reduced to $M/250$) was almost unaffected by $M/100$ α-methyl-glucoside when glucose was used as H-donor, whilst with fructose, partial inhibition was obtained with 10^{-4} M α-methyl-glucoside.

These observations suggested that the action of α-methyl-glucoside was upon reactions leading to production by the cells of supplies of energy, rather than upon some reaction specific to the process of enzyme induction. An inhibitory action of α-methyl-glucoside upon carbohydrate metabolism (O_2 uptake) has actually been discovered several years ago by JOHNSON[4,5,6] who also found that on continued incubation there was, in general, a release from the inhibition ("escape phenomenon") at the lowest concentrations of inhibitor employed. We have repeated and confirmed this observation (at concs. of 10^{-4}–10^{-5} M glucoside). The inhibitory action was found for all sugars tested (at a conc. of $M/250$ sugar), with the exceptions of glucose, gluconate and xylose. However, when glucose was made available at very limited rates (by the action of a preparation of β-galactosidase on lactose[7]) to cells of a *galactose-negative* and *lactose-negative* mutant strain, similar phenomena could be observed with glucose as substrate. The available evidence, including some preliminary experiments with cell-free extracts, suggests that α-methyl-glucoside *may* be a competitive inhibitor of the phospho-fructokinase reaction, but further analysis of the site(s) of action of this inhibitor has been prevented by lack of suitable experimental material.

It seems possible that the mechanism of inhibition of enzymic adaptation by α-methyl-glucoside proposed above is also responsible for the inhibition of adaptation to maltose metabolism observed recently with *α-methyl-glucoside-negative* strains of yeast[8], rather than the postulated inhibition by a substrate analogue. Inhibitions of carbohydrate metabolism by α-methyl-glucoside similar to those reported by JOHNSON[4,5,6] have not only been found with *E. coli* (above) but also with strains of yeast capable of metabolising the glucoside[9,10].

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Received March 6th, 1953

THE RELATION BETWEEN LIPID AND POLYSACCHARIDE CONTENTS OF *BACT. COLI*

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STEPHENSON AND WHETHAM[1] showed that the addition of acetate to an inorganic salt medium increased the lipid content of the *Timothy grass bacillus* but did not affect protein formation; glucose additions increased both protein and lipid contents, the former more than the latter. DAGLEY AND DAWES[2] demonstrated increased polysaccharide contents for *Bact. coli* when glucose and other sugars were added to the growth medium. We have estimated both lipid and polysaccharide in the same

batches of *Bact. coli*, harvested from media containing varying amounts of glucose and acetate 90 minutes after the end of the logarithmic phase when, from earlier work[2] which we confirm, polysaccharide storage is maximal. Polysaccharide contents were determined as previously described[2] using the estimation of SOMOGYI[3] for reducing sugars after hydrolysis with 2 N H_2SO_4. Weighed samples of freeze-dried cells were extracted with a mixture of equal volumes of ethanol and ether in a Soxhlet apparatus overnight and the amount of lipid calculated from the loss in weight of the cells after removal of solvent on a water bath and desiccation to constant weight. Excellent agreement between duplicates was obtained for cells of various lipid contents, and determinations of polysaccharide and protein (micro-Kjeldahl) on the extracted cells gave values agreeing, within 1 %, with those obtained before extraction.

Bact. coli, N.C.T.C. strain 5928, was grown with aeration in media containing 5.4 g KH_2PO_4, 1.2 g $(NH_4)_2SO_4$ and 0.4 g $MgSO_4.7H_2O$ per litre adjusted to pH 7.0 with NaOH; the carbon source was added to give a final concentration of 0.05 M. A series of 2 litre lots of acetate medium to which graded additions of glucose were made was inoculated with acetate-grown cells, nd a similar series of glucose media containing acetate additions inoculated with glucose-grown cells. Polysaccharide

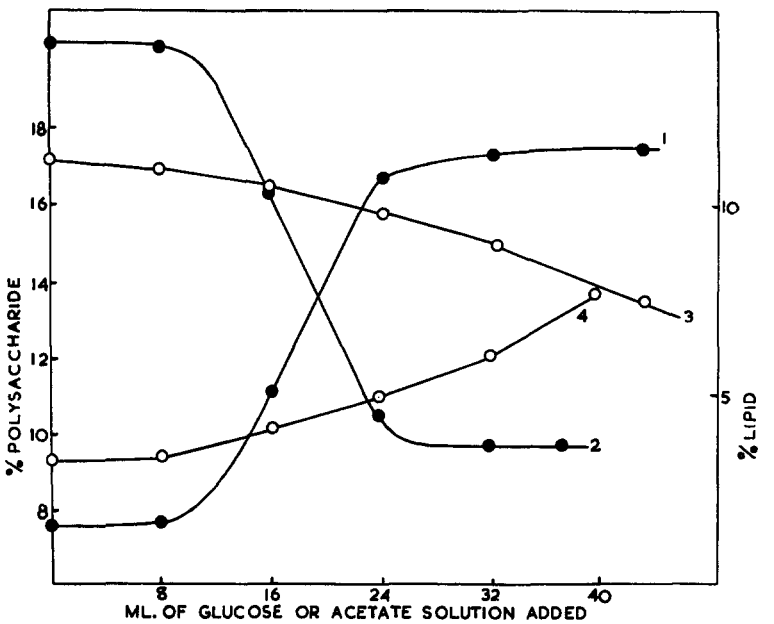

Fig. 1. Blacked-in circles, cells grown in acetate media (2 litres) to which additions of 0.33 M glucose solution were made. Curve 1, polysaccharide content; curve 2, lipide content. Open circles, cells grown in glucose media (2 litres) to which additions of 0.33 M acetate solution were made. Curve 3, polysaccharide content; curve 4, lipide content.

and lipid contents were determined for the fully-grown cultures and for inocula, and results are shown in Fig. 1. The increase in polysaccharide provided by added glucose accompanies a fall in lipid content; some additional lipid storage resulting from the presence of acetate in glucose media is associated with a decrease in polysaccharide.

The following values for maximum polysaccharide content (% dry weight cells) were obtained for a series of carbon sources: glucose, aerated medium 17.3; glucose, unaerated medium 18.6; lactate, 16.3; succinate, 15.2; fumarate, 14.8; malate 14.5; acetate, 7.6.

We wish to acknowledge a grant from the Medical Research Council.

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Received March 26th, 1953