were carried out simultaneously. All measurements were made at 30° C. The results are illustrated in the accompanying graphs.

Curve 1 shows the relation between the pH of sodium hypochlorite solutions and the oxidation potentials. Curve 4 shows the relation between the pH and the reduction potentials of ferrous hydroxide on cotton. The relation of the difference between the oxidation potentials of sodium hypochlorite to the reduction potentials of ferrous hydroxide and the pH is shown in curve 2, and the relation between oxygen uptake by cotton impregnated with ferrous hydroxide and the pH of hypochlorite solution is shown in Curve 3. It is interesting to see the close resemblance in the shape of curves 2 and 3, and the coincidence of the pH for maximum oxygen uptake and the pH for the maximum difference in the potentials with the break in the pH-reduction potential curve. The results will be discussed in detail elsewhere.

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Inhibition of Catechol Bacteriostasis by Molybdates

LARGE areas in and near Somerset are unsuitable for grazing because they cause severe diarrhoea in cattle. These areas, called 'teart', are found on the argillaceous Lower Lias. The disease has been shown¹ to be due to an excess of soluble molybdates in the herbage and the soil; it can be cured by adding copper salts to the diet. A possible explanation of the effect of molybdates is suggested by the following observations.

Molybdates form red complexes with catechols^{2,3}. These are not very stable, and we have found that the colour disappears on warming, or on shaking with ether to remove catechol. Now it is likely (though unproved) that catechols occur in the intestine of ruminants, because they are present in many fodder plants⁴ and because the tyrosine of proteins is converted by intestinal bacteria to phenol⁵ which, in the presence of suitable plant⁶ or animal⁷ oxidases, is oxidized to catechol.

Catechol, like other phenols, is bacteriostatic, but might be less so in the presence of molybdate owing to the formation of the complex. We have found that, in fact, potassium molybdate does reduce the bacteriostatic action of catechol when added in concentrations which by themselves have no effect on the bacteria. In a serial dilution test at 25° C., a catechol concentration of $M/1280$ was needed to inhibit the growth of *Staphylococcus aureus*: when half a mole of potassium molybdate was added per mole of catechol, the concentration of catechol needed to inhibit growth was raised to $M/160$. Even $M/20$ potassium molybdate was without effect on *S. aureus*. With *Escherichia coli* similar results were found; the minimum bacteriostatic concentration of catechol was raised from $M/160$ to $M/40$ by adding

half a mole of potassium molybdate per mole of catechol used.

Phenol, *o*-cresol and resorcinol form no coloured complexes with potassium molybdate; correspondingly, we found that it had no effect on their minimum bacteriostatic concentrations.

These biological results suggested that a complex, $K_2MoO_4 \cdot 2C_6H_5O_2$, was formed, and a colorimetric experiment was made at 25° C. to test this theory.

Molarity in aqueous solution		Colour intensity in arbitrary units
Potassium molybdate	Catechol	
0.06	0.02	1.00
0.533	0.0266	1.29
0.04	0.04	1.84
0.0266	0.0533	2.27
0.02	0.06	1.77

The total molarity of solute is kept constant, and the colour intensity, and hence presumably the concentration of the complex, is a maximum at a proportion equivalent to $K_2MoO_4 \cdot 2C_6H_5O_2$.

These facts are compatible with the theory that the activity of bacteria in the normal intestine is controlled by catechols: that molybdates reduce the effective concentration of catechols by forming complexes: and that with the removal of the control, bacterial activity becomes excessive and causes diarrhoea. It is realized that further investigation would be needed to confirm this theory; in particular, the effect of copper as an antidote still requires explanation.

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I AM grateful to Messrs. McGowan and Brian for allowing me to read their letter and to add my comments. Some data obtained in 1943, when working in the Unit of Animal Physiology at Cambridge, are of interest in connexion with their observations. It was found that fresh aqueous plant extracts give a colour reaction when molybdate is added. This reaction is due to the presence of substances, usually classified as 'tannins', which contain vicinal phenolic hydroxyl groups. Fresh grass cuttings were fed for two days to a rumen-fistula sheep; the rumen contents were then removed through the fistula. An extract of the ingesta thus obtained gave a colour reaction characteristic of catechols, with both ammonium molybdate and ferric chloride. This shows that the reaction between molybdate and catechols suggested by McGowan and Brian does, in fact, occur. Their explanation still remains to be tested by experiments on animals, but the readiness of molybdates to form organic complexes must be an important factor in the etiology of molybdate poisoning in cattle.

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