SHORT COMMUNICATIONS

EFFECT OF SODIUM-1-NAPHTHYL PHOSPHATE ON THE ELECTRO-DEPOSITION OF COPPER

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IT is perplexing that small variations in the structure of an additional agent can change its brightening ability and also its effectiveness in plating solution (*e.g.* mercaptoethanol and dithiothreitol)[1,2]. However, a detailed study of the role of the functional groups in the overall performance of the addition agent is inadequate. The present authors started a systematic study on the role of the structure of the organic compounds on copper electrodeposits on copper single crystals.

The effect of sodium-1-naphthyl phosphate on the morphological and electrochemical behaviour of the (111) plane of copper single crystals from a solution of acid copper sulphate containing sodium-1-naphthyl phosphate at various current densities was undertaken to find out whether the additive causes hard facing, resulting in greater corrosion resistance of the material.

The experimental procedure has been described in detail elsewhere[3]. An atomically smooth (111) face of copper (supplied by Monocrystals) was prepared by electropolishing in phosphoric acid at a cell potential of 1.2 V[4]. An electrolytic bath of composition 0.25 mol dm^{-3} $CuSO_4$ in 0.1 mol dm^{-3} H_2SO_4 was prepared and sodium-1-naphthyl phosphate (SNP) of the desired concentration was used. Copper deposits of thickness 3.6 μ were deposited at a given current density. The surface appearance of the copper electrodeposit was examined under a metallurgical microscope.

The deposit was scraped when higher concentrations of SNP (10^{-4} mol dm^{-3}) were added to the acid copper sulphate and studied using IR spectrometry.

For purposes of comparison, the effect of H_3PO_4, β-naphthol and naphthyl acetate was also undertaken and it was found that SNP gives better results at lower concentrations. From the results it appears that the concentration of SNP required to bring about the modification of the growth habit depends upon the CD and the critical concentration.

As noticed by earlier workers[5] (figure 1) at 2 and 4 mA cm^{-2}, the triangular pyramids and at 5, 7.5, 10 and 12.5 mA cm^{-2}, the hexagonal blocks were observed. At 2 mA cm^{-2} the triangular type of growth gets truncated in the presence of 10^{-6} mol dm^{-3} of SNP and this changes to a levelled fine-grained polycrystalline deposit at 10^{-5} mol dm^{-3} (figure 2). At 5 mA cm^{-2} in pure solution the characteristic growth changes to a very fine grained polycrystalline deposit at a concentration of 10^{-4} mol dm^{-3} of SNP. However,

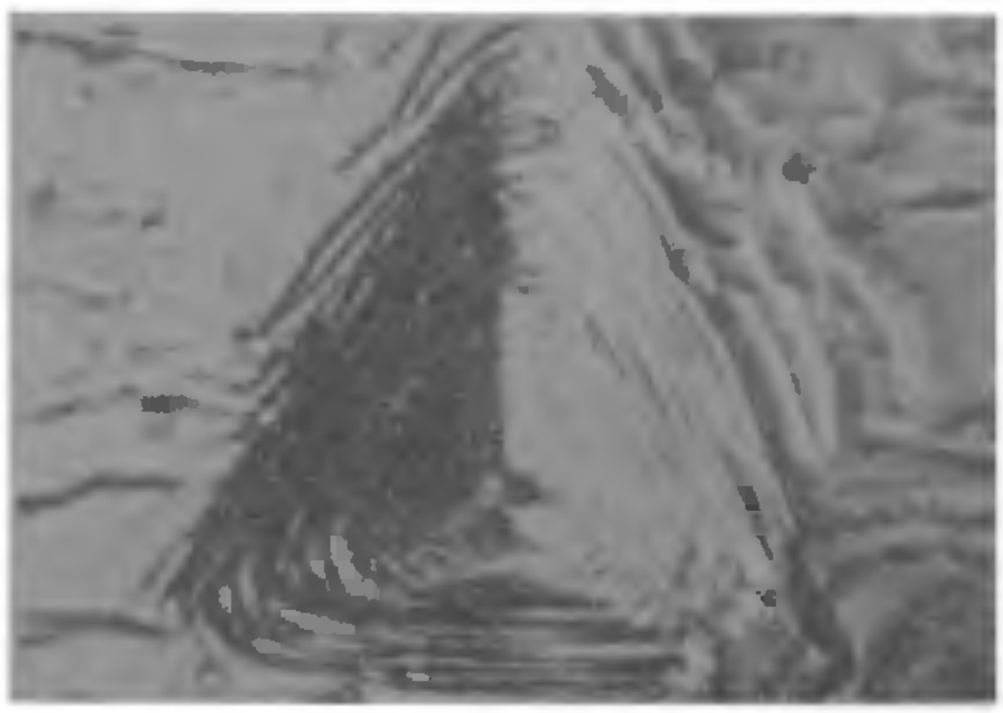

Figure 1. Pyramidal growth of copper deposited on Cu (111) from an acid copper sulphate bath at 2 mA cm^{-2} (625 ×)

Figure 2. Levelled fine grained polycrystalline deposit in the presence of 10^{-5} mol dm^{-3} of SNP at 2 mA cm^{-2} in acid copper sulphate (625 ×)

the critical concentration of SNP needed to bring the above growth habit modification depends on the current density. The η-log i relationship is linear in pure solution with a value of 120 ± 10 mV and 10^{-3} mol dm^{-3} of SNP with a value of 90 ± 10 mV.

It is interesting to note that the levelled fine grained deposit was obtained with much lower concentration of SNP than with phosphoric acid and β-naphthol. This indicates that the electrochemical discharge reaction may occur through complex formation, which is also supported by the IR data of the scraped deposit. Further, the deposit exhibits greater corrosion resistivity when it is subjected to standard corrosion monitering technique.

It could thus be concluded that SNP appears to be a better additive than the conventional additives. The mechanism of habit modification is under investigation.

9 April 1985

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GROWTH INHIBITION OF *ENTAMOEBA HISTOLYTICA* BY METHYLGLYOXAL BISGUANYL HYDRAZONE (MGBG)

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ALIPHATIC polyamines occur ubiquitously in living organisms where they accompany and possibly regulate biosynthesis of informational macromolecules[1-3]; prokaryotes contain putrescine and spermidine and eukaryotes contain spermine as well[1]. Protozoa and certain lower eukaryotes and fungi lack spermine[4,5] and the growth of protozoa have been shown to be related to polyamine biosynthesis[6]; inhibitors of polyamine biosynthesis like difluoromethylornithine (DFMO) cure certain experimental protozoal infections caused by *Trypanosoma brucei* and *Eimeria tennella*[7,8]. DFMO has been found to inhibit exoerythrocytic schizogony of *Plasmodium berghei* but had no effect on the erythrocytic schizogony[9]; polyamine metabolism has also been implicated in the mechanism of chloroquine action[10]. *Acanthamoeba culbertsoni* was found to contain spermidine and putrescine but lacked spermine[11,12]. *Entamoeba invadens* contained high concentration of putrescine and significant amounts of spermidine and spermine[13] but *E. histolytica* has putrescine and spermidine; spermine was detected only in traces[12]. Ferrante *et al*[14] recently reported growth inhibition of *E. histolytica* by synefungin, an inhibitor of transmethylation reaction and possibly also polyamine metabolism[15]. Gillin *et al*[16] recently reported growth inhibition of *Giardia lamblia* by DFMO but this compound was ineffective on *E. histolytica*. Our results on the growth inhibition of *E. histolytica in vitro* by MGBG, an inhibitor of S-adenosylmethionine decarboxylase, a key enzyme of polyamine biosynthesis, support that polyamine metabolism may be a chemotherapeutic target for antiamoebic chemotherapy.

An axenic culture of *E. histolytica* NIH-200 was grown in Diamond's TPS-1 monophasic medium[17] as modified by Imam by incorporating RNA (2.5 mg/ml). α-Methyl ornithine and MGBG (Sigma Chemical Co., U.S.A.) were obtained through kind courtesy of Prof. Yogesh Awasthi, Texas Medical School, Galvestan Texas. The inhibitors were dissolved in TPS-1 medium, sterilized by filtration through millipore filters (0.22 μm) and added to 10 ml cultures contained in screw capped tubes to get the desired concentration; tubes were incubated at 37° C. At desired intervals, the tubes were chilled to dislodge the amoebae adsorbed to surface of tubes, mixed and counted by hemocytometer.

Results on the growth inhibition of *E. histolytica* by MGBG are presented in figure 1. The normal growth of amoebae occurred with a lag of about 24 hr and proceeded rapidly reaching the maximum value around 96 hr and declined thereafter. The growth was strongly inhibited at 1- and 2.5 mM concentration of MGBG; complete growth inhibition was observed at 5 mM MGBG (figure 1). α-Methylornithine permitted considerable growth of *E. histolytica*; 4.95 million amoebae/10 ml were detected at 96 hr growth in the presence of 1 mM α-methylornithine as compared to 7.35 million in control tubes. Increase in the concentration of this inhibitor to 2.5 or 5 mM did not further inhibit the growth (table 1). The poor inhibition of