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.

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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

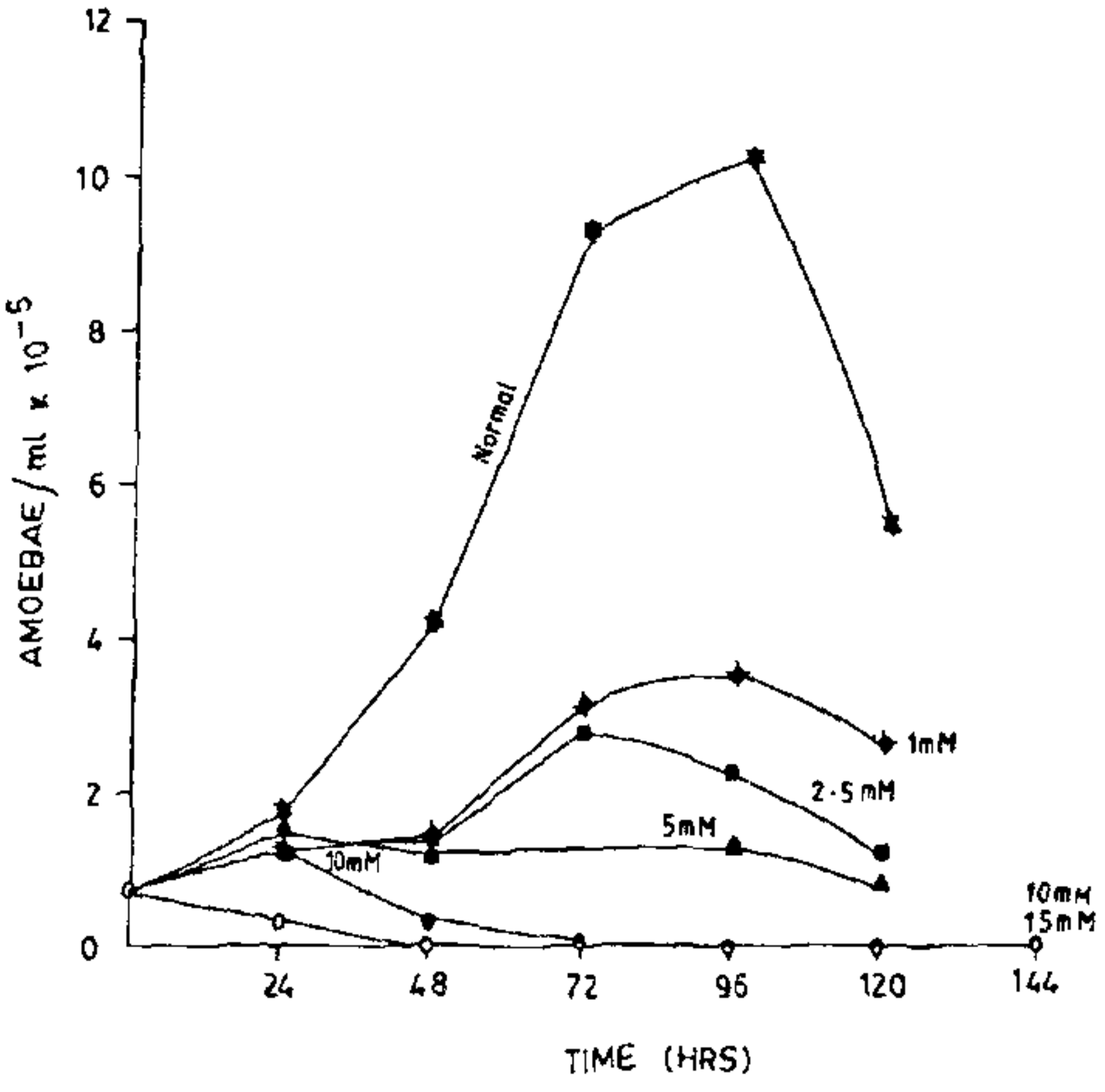

Figure 1. Effect of methylglyoxalbisguanylhydrazone on growth of *E. histolytica.*

Table 1 *Effect of α-methylornithine on the growth of E. histolytica.*

Conc. methyl-ornithine (mM)	Amoebae/ml × 10^{-5}			
	24 hr	48 hr	72 hr	96 hr
1.0	+	++	+++	4.95
2.5	+	++	+++	4.95
5.0	+	++	+++	4.20
Control	1.55	2.00	6.94	7.35

amoebic growth by α-methylornithine may be due to the relative insensitivity of amoebic ornithine decarboxylase to this inhibitor or due to the presence of alternate pathway for putrescine biosynthesis. Gillin *et al*[16] observed that growth of *E. histolytica* could not be inhibited by 20 mM DFMO. Ornithine decarboxylase of *E. histolytica* may thus differ from the enzyme from other protozoal systems which are highly sensitive to α-methylornithine. On the other hand, inhibition of amoebic growth by MGBG indicates the presence of a S-adenosylmethionine decarboxylase that is sensitive to this compound. The inhibition of growth of *E. histolytica* by synefungin[15] may also be directed at this site, and analogs of these inhibitors may serve as alternative chemotherapeutic agents.

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DINITROGEN FIXATION IN PEARL MILLET

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PEARL millet is an important grain and fodder crop grown in northern India during summer, generally with low levels of nitrogen fertilization. It is reported that this crop harbours a variety of nitrogen-fixing bacteria in its rhizosphere and rhizoplane[1,2] which may provide a substantial part of its nitrogen requirement. An attempt was, therefore, made to determine the extent of nitrogen fixation in this crop and