

electron spin resonance signal was observed in all cases. An example is shown in Fig. 1. If the sample was kept in a dry atmosphere, the signal did not decrease appreciably in a week. The decay of resonances reported by other workers<sup>6</sup> may have been due to the presence of moisture in the gases used, an effect which has been observed in some biological materials<sup>7</sup>. When moisture was present in our experiments at the time of irradiation, however, we were unable to detect any electron spin resonance signal even with a dose of  $10^8$  rads.

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### Protective Effect of $\beta$ -Aminoethylthiosulphuric Acid against Ionizing Radiation

IT was recently demonstrated that thiosulphate esters rapidly react with protein sulphhydryl groups to give mixed disulphides<sup>1</sup>. As Eldjarn and Pihl<sup>2</sup> have attributed the protective properties against radiation of certain sulphur compounds (for example, cysteamine, cystamine) to their ability to form mixed disulphides with active sulphhydryl or disulphide groups in the cell, the possibility was considered that thiosulphate esters may be active protectors against ionizing radiation.

This has now been verified with  $\beta$ -aminoethylthiosulphuric acid<sup>3</sup>, and the protecting effect of this compound has been compared with that given by cysteamine. The acute toxicity of  $\beta$ -aminoethylthiosulphuric acid (determined as  $LD_{50}$  by intraperitoneal injection into 50 albino mice according to Miller and Tainter<sup>4</sup>) was found to be  $0.90 \pm 0.02$  gm./kgm. body-weight, whereas the  $LD_{50}$  of cysteamine hydrochloride determined by the same technique was obtained as  $0.50 \pm 0.01$  gm./kgm. body-weight. Cysteamine hydrochloride is then about 2.4 times as toxic as  $\beta$ -aminoethylthiosulphuric acid, calculated on an equimolar basis.

The protective effect of  $\beta$ -aminoethylthiosulphuric acid on mice, irradiated with a lethal dose of X-rays, as compared with that of cysteamine hydrochloride, is shown in Fig. 1 (each compound was injected

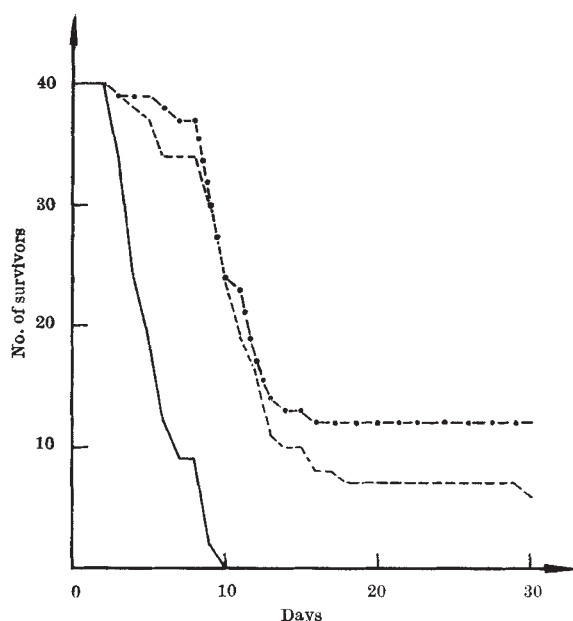


Fig. 1. Protective effect of  $\beta$ -aminoethylthiosulphuric acid and cysteamine against X-rays. Male albino mice exposed to 1,000 r. Protective compounds injected intraperitoneally 15 min. before irradiation. Without protector, —;  $\beta$ -aminoethylthiosulphuric acid, 0.45 gm./kgm. body-weight, - - -; cysteamine hydrochloride, 0.25 gm./kgm. body-weight, - · - · -.

at a dose corresponding to 0.5  $LD_{50}$ ). It is evident that  $\beta$ -aminoethylthiosulphuric acid increases both the time and rate of survival, although the latter showed a smaller increase than in mice injected with cysteamine. The possibility that the protection given by  $\beta$ -aminoethylthiosulphuric acid is due to cysteamine or cystamine, formed from  $\beta$ -aminoethylthiosulphuric acid in the body, cannot be excluded, but appears unlikely from previous *in vitro* studies on  $\beta$ -aminoethylthiosulphuric acid<sup>1</sup>.

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### Leucocytosis in Response to Bacteraemia as a Feature of the Acute Radiation Syndrome in the Plaice

BACTERIAL invasion of the blood-stream is a marked feature of the acute radiation syndrome in the mammal following exposure to mid-lethal and supra-lethal doses of ionizing radiation<sup>1-6</sup>. It is generally considered that the bacteria are enteric in origin and that they gain access to the blood-stream through failure of the host defence mechanism, following the radiation insult, that is, ulceration of the gastro-intestinal tract, and leucopenia<sup>1,7</sup>. According to Bennett *et al.*<sup>8</sup> death attributable to such infection of the blood-stream in dogs is always associated with leucopenia, and Cronkite and Brecher<sup>6</sup> consider that the body of evidence available appears to establish