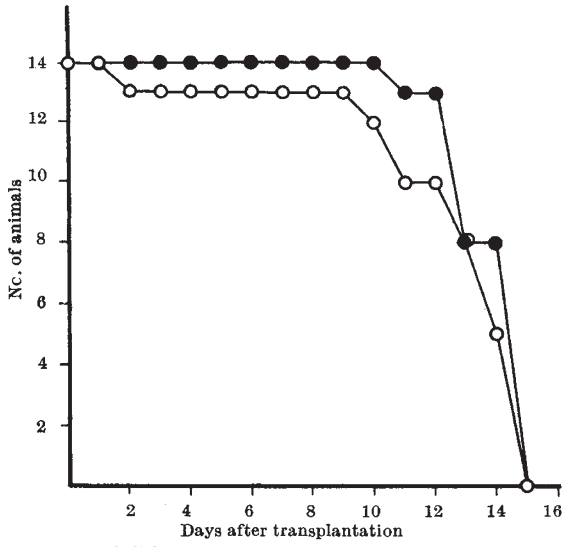

Fig. 3. Ehrlich ascites sarcoma: survival time of hydrogen peroxide – treated (—●—) and control (—○—) mice

hydrogen peroxide (Fig. 2), yet the treatment had no influence on the survival time of these mice (Fig. 3).

From this work it is not possible to assess the activity of hydrogen peroxide *per se* against tumour growth, because of some uncontrolled experimental conditions. In particular, the intake of food and water must be taken into consideration. We noticed that the intake of hydrogen peroxide solution by treated animals was much smaller than the intake of distilled water by control animals. Diminished food intake followed by loss of weight in healthy rats drinking hydrogen peroxide as compared with rats supplied with tap water was observed by Ghadially and Wiseman[3]. It seems very probable that changes in nutritional conditions brought about by drinking hydrogen peroxide (diminished food intake, or direct action of hydrogen peroxide on some essential constituents of ingested food) may be causal factors sometimes mediating an inhibitory effect on certain kinds of tumours.

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Anticholesterol Activity of α-Lipoic Acid

A recent report in *Nature*[1] concerning the 'anticholesterol' properties of α-lipoic acid has prompted communication of similar experiments in which different results were obtained.

In our work two groups of male Dutch belted rabbits (*A* and *B*) were maintained on a diet containing 2 per cent cholesterol and 6 per cent corn oil for two months. A third group (*C*) was kept as a normal control. One of these groups (*B*) also received daily intraperitoneal injections of 1 mgm. of α-lipoic acid, which represented 0·5–1 mgm./kgm. body-weight for the animals used. After eight weeks the animals were killed, the sera assayed chemically for cholesterol[2] and ultracentrifugally for β-lipoproteins[3] and the aortas graded visually (on a 0–4 scale) for atheromata. The results are shown in Table 1.

Table 1. Autopsy Results for Rabbits fed with Cholesterol

Group	No.	Atheromata (average) Arch	Thoracic	Liver weight (gm.)	Gain in weight (gm.)
A	10	2·10	1·25	93	219
B	10	2·50	1·65	90	49
C	5	0·00	0·10	63	449

Table 2. Serum of Rabbits fed with Cholesterol

Group	Average cholesterol (mgm./100 ml.)	Average lipoproteins (mgm./100 ml.) *Sf* 0–11	12–20	21–35	35–100	100–400
A	1,893	220	517	839	997	663
B	1,853	263	731	1,015	782	443
C	175	146	75	43	30	25

The most obvious source of discrepancy is the fact that Angelucci and Mascitelli-Coreandoli[1] administered considerably more α-lipoic acid than we did. In the absence of weights of the animals, it cannot be said if their rabbits showed even less weight gain than ours did. It is also worth noting that whereas their lipoic-acid group showed 20 per cent of the serum cholesterol-levels of the cholesterol-fed controls, they showed 66 per cent of the aorta cholesterol. In our work there was increased atherosclerosis in the lipoic acid group.

These results, indicating one of the lower limits at which no effect of α-lipoic is discernible, may serve as a guide in defining dosages, and may stimulate further work.

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The discrepancy between our results and those of Kritchevsky and Moyer could be explained as follows:

(*a*) In comparison with the dosages used by Kritchevsky and Moyer, our rabbits were given a smaller total amount of cholesterol and a greater total amount of α-lipoic acid, with a ratio between cholesterol and α-lipoic acid comparatively in favour of the latter in unit time.

(*b*) That there is a critical value in the α-lipoic acid dosage for the 'anticholesterol properties' can be argued from the fact that in humans with high serum cholesterol-levels, chronic treatment with α-lipoic acid 25 mgm. twice a day did not change the serum cholesterol-levels, whereas 50 mgm. once a day gave a moderate decrease in the serum cholesterol levels (Consolo *et al.*, *Boll. Soc. Ital. Biol. Sper.*, 32, 1325; 1957).

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