was marked. Congestion and oedema was also observed in the lungs. The hilus of the kidney had gelatenous changes. Histopathological changes were mainly predominant in the liver, kidney and the intestines in both the species. It varied from mild degenerative change in hepatic parenchyma to that of severe hepatic change tending towards necrosis. Kidney revealed extensive haemorrhagic zones at the tubular areas with varied degrees of regressive changes comprising hyaline changes in the tubules and the constricted glomerular tuft. Lesions in the abomasum varied from slight erosion of the epithelial cells to that of haemorrhagic zones on mucosa and submucosa. Intestine also showed severe congestion and erosion of mucous membrane.

On discontinuation of Parthenium after 30 days, the remaining animals which were showing signs of toxicity gained in health. Repigmentation occurred after 3 weeks in buffalo bull calves showing signs of recovery from dermatitis. The ulcerations developed on the muzzle and lips of the surviving cross-bred bull calves showed marked signs of regeneration.

It is not surprising the Parthenium hysterophorus L. is poisonous to animals. Some members of the compositae family to which it belongs, are known to be toxic when eaten by live-stock[6]. Ingestion of Helinium microcephalum, a weed that grows in many parts of Texas, USA, and Mexico was observed to produce acute poisoning and death in cattle, sheep and goats[7]. Toxic properties of this weed have been attributed to helenalin, a sesquiterpene lactone[8]. Similarly tenulin, the major sesquiterpene lactone constituent of Helinium amarum was found to be toxic and was responsible for milk bittering[9]. It remains to be investigated whether the sesquiterpene lactone parthenin from Parthenium hysterophorus L., which has been attributed to be responsible for allergic contact dermatitis in man[10] is responsible for acute illness and death in cattle. Unless effectively checked, the aggressive spread of Parthenium in India may likely to pose considerable danger to live-stock, particularly under drought conditions.

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Influence of oral glucose feeding on endotoxin lethality in mice[1]

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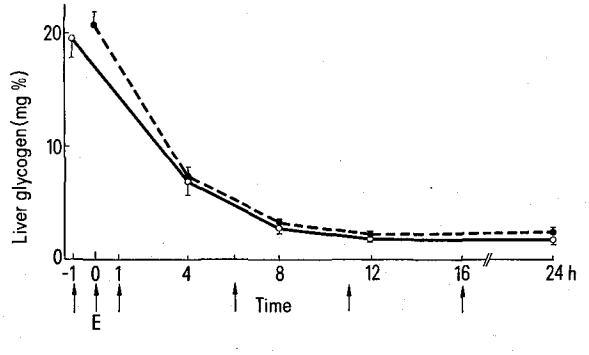
Summary. Prolonged feeding of physiological solutions of glucose (5%) by gavage did not protect against either endotoxin death or liver glycogen depletion in mice.

It is well-known from clinical and experimental observations that one of the most characteristic symptoms of endotoxicosis is the imbalance of carbohydrate metabolism, and many experimental data suggest that loss of carbohydrate reserves may be directly related to the severity of endotoxicosis and thus influence the survival rates[4,5]. If death is directly related to endotoxin-induced carbohydrate loss, it would be logical to assume that survival would be improved by administration of glucose. Several groups of experiments indicate, however, that the administration of exogenous glucose i.v. or i.p., to mice or guinea-pigs, made hyperreactive to endotoxin by BCG[4,5] or CCl_4[6], respectively, or during endotoxin shock in dogs[7], prolongs life but has little effect on the ultimate mortality. Furthermore, significant protection against endotoxin toxicity is not obtained during alloxan-induced diabetes in BCG-sensitized mice, in spite of the fact that diabetes is associated not only with hyperglycemia but also with decreased glucose utilization and with increased gluconeogenesis[5].

The experiments reported below are different from earlier studies at least in three respects: a) since the physiological and pathological reactions of animals rendered hyperreactive to endotoxin are different from normal animals in many aspects[4,5], we studied the effect of exogenous glucose on endotoxin lethality in normal mice; b) glucose was administered by stomach tube in the hope that it would reach the liver directly via the portal venous



Influence of glucose load on liver carbohydrate levels. Mice were injected with endotoxin at time 0 and given an oral glucose (●) or water (○) load at the arrow. Glycogen determination was carried out on liver biopsies taken just before 1st and 2nd feedings and at the indicated times thereafter. All values are an average of 8 separate determinations ± SE.

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system, since the liver plays an important role in endotoxicosis[8] and endotoxin depletes carbohydrate reserves first of all from the liver[4], and reverses gluconeogenesis; c) glucose feeding was prolonged until the 16th hour after endotoxin challenge, contrary to other studies cited above with only 1 or 2 injections and small quantities of glucose.

Materials and methods. Swiss, male, albino mice (24 ± 2 g) were housed on laboratory food and water ad libitum; food was withdrawn at the beginning of the experiments. To determine lethality, mice were injected with 500 µg of E. coli 026:B6 endotoxin (Difco, lot 586787) i.p. and the survival was recorded 48 h later. The LD_{50} was calculated according to Reed and Muench[9]. Glucose (5%), sodium chloride (0.9%), or water, in 0.5 ml, was given by stomach tube 1 h before and 1, 6, 11 and 16 h after endotoxin challenge.

Liver glycogen levels were determined according to the method of Kemp and Kits van Heijningen[10] as adapted previously[11]. The glycogen level is expressed as mg/100 mg fresh liver weight. The results were evaluated by the Student t-test and the chi square test. A SE was calculated for all mean values.

Results and discussion. Data in the table show that the administration of a total of 125 mg glucose (5% solution) by gavage (25 mg per dose, 1 h before and 1, 6, 11, 16 h after endotoxin challenge) did not alter endotoxin lethality. Oral administration of physiological saline was also without effect on the outcome of endotoxin lethality. There was 36%, 40.7% and 40% survival level in the water, physiological saline and 5% glucose treated groups, respectively.

Influence of glucose or saline feeding on endotoxin lethality in normal mice

Treatment	Living/Total (48 h)	Percent survival	Statistics
1. Water	9/25	36	
2. Physiological saline	11/27	40	2 VS 1 NS
3. 5% glucose	10/25	40	3 VS 1 NS

NS = not significant.

Data in the figure clearly show that liver glycogen both in the water-treated and physiological glucose-treated groups was significantly lower within 4 h after endotoxin challenge, and continued so until a minimal carbohydrate level was achieved in both groups. The liver glycogen level did not increase in response to glucose feeding in endotoxin-challenged groups.

These results support earlier observations[4-6] obtained in endotoxin hyperreactive animals, with i.v. glucose administration, that exogenous glucose supply does not influence the outcome of endotoxin lethality. As in earlier studies[4,5] with parenteral glucose administration, exogenous glucose given by stomach tube also failed to prevent glycogen depletion during endotoxicosis (figure), either because it was metabolized and/or converted to muscle glycogen since mucsle glucogenesis is not impaired in endointoxicated animals[4]. It may be that liver glycogenesis is controlled at 2 levels; glucose-induced cycle can only proceed under normal conditions but cortisone-induced phase can proceed just as well in endotoxin poisoned animals as in normal mice, although cortisone-sparing of liver glycogen levels could only be secondary to protection against endotoxin lethality. Finally, the primary event in endotoxin death still remains elusive.

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Influence of anaesthesia by carbon dioxide and ether on locomotor activity in *Drosophila melanogaster*

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Summary. Locomotor activity of Drosophila melanogaster was investigated in 2 recently caught unrelated wild stocks. It is strongly affected by anaesthesia with either carbon dioxide or ether. Both anaesthetics had opposite effects. Ether produces a longlasting decrease in activity. Carbondioxide causes an increase of locomotor activity, but smaller and of shorter duration than the effect of ether.

Research on activity of Drosophila has shown that, it is a complex character, which is under control of both genetic and environmental factors[1-5]. It could be expected that anaesthetics, used to immobilize flies, would affect the locomotor activity in later life. During experiments on locomotor activity, we obtained evidence that these effects could be considerable.

Therefore an experiment was started to investigate the long-term effects of both ether and CO_2 on this character. The locomotor activity of 2 wild strains of Drosophila melanogaster, recently collected in Dahomey (now Benin) and Spain (La Mancha) was measured in an apparatus that was especially constructed for this purpose[6]. It is a modification of the apparatus, used by Ewing[1], and consists of a row of 20 vials, which can be transversed in one direction only.

The mean activity score was calculated as the mean number of vials the flies have visited after 20 min. The activity tests were performed at 20 ± 1 °C. Virgin flies were collected with either ether or CO_2 and tested at ages of 30, 300 and 450 h. Some of the flies of the 300 and 450 h old flies were submitted to a subsequent anaesthesia with either CO_2 or ether. The ether was tested on the presence of peroxides. The results were submitted to an analysis of variance. No interactions between the first

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