Glucagon Treatment of Muscular Dystrophic Mice: A Lack of Effect

POPE[1] reported that glucagon, at doses from 2 to 20 μg/mouse/day, increased the mean survival time, the mean maximum weight and the age at maximum weight of genetic muscular dystrophic mice. These treatments also improved the clinical condition of the mice. Based on POPE'S initial report, BRADLEY et al.[2] attempted to confirm POPE'S observations but did not succeed. In this communication, we wish to report that our results support the conclusions of BRADLEY et al.[2]. At the concentrations prescribed by POPE, glucagon had no observable effect on genetic muscular dystrophic mice.

Thirty-seven male and female muscular dystrophic mice (*dy/dy*) of the 129/ReJ strain were purchased from the Jackson Laboratory, Bar Harbor, Maine. They were divided into 3 treatment groups as shown in Table I. To avoid a cage to cage variation, 3 mice, 1 from each treatment group identified by ear mark, were housed in 1 transparent plastic cage. The extra mice on 10 μg glucagon were also housed 3 to a cage. Purina Laboratory Chow and water were available ad libitum. Lights in the mouse room were kept on from 06.00 to 18.00 h.

Glucagon (Lilly) was given s.c. once a day in a volume of 0.1 ml diluent at 2 or 10 μg per mouse, 7 days a week. The experiment was started when the mice were 52–56 days of age and was terminated 100 days later. At that time, the dystrophy of the surviving mice was quite severe and they had not been gaining weight for several weeks. The dystrophic condition of all the mice was confirmed by pathological examination upon autopsy.

Table I presents the mean maximum weight attained by each group of mice. Male mice were significantly heavier than female mice although the difference was only 2 g. Our mice, both male and female, were lighter than POPE'S[1] probably because of the difference in diets. POPE used Purina Mouse Breeder Chow which has a higher energy content and fat content than the Purina Laboratory Chow we used. In our experiment, there was no difference in body weight within each sex between either of the glucagon-treated groups and the diluent group.

Table II presents the number of experimental days survived by each mouse. The female mice as a group survived longer than the male mice, a finding also reflected in POPE'S data[1]. In our study, no difference was observed within each sex between either of the glucagon-treated groups and the diluent group. We, therefore, cannot substantiate POPE'S claim that glucagon treatment is beneficial to the muscular dystrophic mice. Other means must be devised for the treatment of this disease.

Table I. Maximum weight[a] of dystrophic mice during the experiment

	Female (*N*)	Male (*N*)
Diluent[b]	14.0 ± 2.6 (5)	15.6 ± 1.7 (5)
2 μg glucagon	13.0 ± 1.9 (5)	15.6 ± 3.0 (4)
10 μg glucagon	14.0 ± 1.3 (10)	15.8 ± 1.9 (8)
Combined	13.7 ± 1.5 (20)	15.7 ± 2.0 (17)[c]

[a] Mean ± S.D.(g). [b] The diluent (Lilly) contained 1.6% glycerine and 0.2% phenol, pH 2.5–3. [c] Significantly different from the combined female group at $p < 0.005$.

Table II. Number of experimental days survived by each dystrophic mouse

	Survival (days)	$\bar{X}$ ± S.D.
Female mice		
Diluent	46, 100, 100, 100, 100	89.2 ± 24.1
2 μg glucagon	38, 39, 100, 100, 100	75.4 ± 33.7
10 μg glucagon	51, 54, 70, 100, 100 100, 100, 100, 100, 100	87.5 ± 20.7
Combined		84.9 ± 24.4
Male mice		
Diluent	23, 52, 66, 66, 100	61.4 ± 27.8
2 μg glucagon	3, 3, 55, 81	35.5 ± 39.0
10 μg glucagon	10, 19, 37, 62, 95 98, 100, 100	65.1 ± 38.5
Combined		57.1 ± 35.8[a]

The experiment was terminated after 100 days
[a] Significantly different from the combined female group at $p < 0.01$.

Zusammenfassung. Nachweis, dass Körpergewicht, Dystrophie und Sterblichkeit bei männlichen und weiblichen muskeldystrophischen Mäusen (*dy/dy*) nach 100tägiger Behandlung mit Glukagon (2 oder 10 μg pro Tag, s.c.) unverändert blieben.

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[1] R. S. POPE, Am. J. Physiol. *225*, 518 (1973).
[2] W. G. BRADLEY, J. G. POLGAR, M. H. WILLIAMS and H. G. BODDIE, Br. med. J. *3*, 699 (1972).

Presence of Resilin in a Scorpion *Palamnaeus swammerdami* and its Role in the Food-Capturing and Sound-Producing Mechanisms

It is known that the pedipalp of scorpions ends in a chela formed of an immovable tibia and a movable tarsus. The tarsus moves against the strong processes of the tibia for grasping the food[1]. CLOUDSLEY-THOMPSON[2] reports that the scorpions produce sound by briskly drawing the tip of the tarsus backwards and forwards against the teeth of the tibia. In a recent study on the musculature of pedipalp of a scorpion *Heterometrus scaber*, MATHEWS[3] reports that there are only retractor muscles for closing the tibia and extensors are totally lacking, in contrast to the chela of crabs where both types of muscles occur. Inspite of the absence of extensors, the chela in