'active' sugar transport across the mucosal layers of the small intestine requires the presence of oxygen may not necessarily mean that aerobic conditions are essential for the activity of a sugar pump localized in the cell membranes; the effects are equally explicable if anoxia increases the relative leakiness of the mucosal faces of the absorbing cells $(K_1/K_2 \geqslant p_1/p_2)$. In a similar fashion, differences in the relative leakiness of the faces of the two poles of mucosal cells from different regions of rat intestine may underly the differences in the capacity of these regions to absorb either glucose[5,6] or galactose[7]. Now a substrate may possess physical and chemical properties such that it can be operated on by the pumping process p, but these properties need not be the same as those which determine the magnitudes of K_1 and K_2. For example, in a given system, the former might depend on a particular stereochemical configuration of a substrate, while the K-values might depend on the molecular weight. D-xylose has not been shown to be actively transported against a concentration gradient in small intestine, but there is recent evidence suggesting an interaction between the pentose and glucose during absorption[8]. Xylose may share an inwardly directed sugar pump with glucose, but intracellular pumping, even at high rates, does not necessarily mean that transcellular active transport will occur.

In other similar model systems, but in which the processes underlying the substrate leak across the cell faces are more elaborate than those of simple diffusion (for example, facilitated diffusion), competition between substrate molecules for exit from the cell could have important effects. For example, the overall specificity of the whole system for transcellular transport of a particular class of substrates, including such effects as those of one substrate on another, will be the resultant of the specificity of the intracellularly directed pump for substrate and the specificity of the leak.

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Stimulation of Insulin Secretion in vitro by Adenosine Triphosphate

WE have already shown that the metabolites (glucose, citrate, β-hydroxybutyrate), the final function of which is the production of energy in the $\sim P$ form[1,2], give place to release of insulin in vitro when they are added to the incubation buffer surrounding slices of pancreas. For this reason, we considered it interesting to determine if ATP when added to the incubation buffer produces any effect on the release of insulin, although what is generally accepted is that the metabolic processes in which this nucleotide takes part are intracellular.

A piece of rabbit pancreas weighing 200 mg was incubated for 3 min at 37° C in Krebs–Henseleit buffer containing ATP 1×10^{-3} M and another piece of the same weight (± 5 mg) was incubated in the same buffer but containing hydrolysed ATP (1×10^{-3} M–N/10 hydrochloric acid for 15 min at 100° C) as control (final pH 7). The insulin of the buffer was extracted by the method of Grodsky and Tarver[3] and evaluated by the technique using adipose tissue of the rat epididymis. The results

are expressed in uptake of glucose and the statistical study was performed using the paired observations method[4].

Table 1. EVALUATION OF THE INSULIN (GLUCOSE UPTAKE) EXTRACTED FROM THE INCUBATION BUFFER (CONTAINING ATP OR HYDROLYSED ATP) SURROUNDING PIECES OF RABBIT PANCREAS

Metabolites		Mean glucose uptake mg/100 of tissue/4 h
ATP $(1 \times 10^{-3}$ M)	(20)	1·082
		$\pm$ 0·066* $P = 0.01$
ATP hydrolysed $(1 \times 10^{-3}$ M)	(20)	0·781

*S.E. The values in parentheses represent the number of experiments.

As can be seen from Table 1 the insulin extracted from the buffer of incubation containing adenosine triphosphate is higher than that obtained from the control. The difference is highly significant. We think that this fact supports our hypothesis about the role of the ATP in release of insulin.

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Increased Survival-time in Dystrophic Mice treated with Methylandrostenediol Dienanthoylacetate

ANABOLIC steroids have been noted to increase survival time in dystrophic mice, Jax/129 (dydy), but the disease syndrome was not otherwise altered. Testosterone propionate did not possess this ability[1,2]. It was of interest to evaluate an anabolic steroid, 17-α-methylandrostene-5-diol-3β-17β-dienanthoylacetate (MADD), which has a prolonged period of activity.

Mice with hereditary muscular dystrophy (dydy) were obtained by mating Jax/129 mice (Dydy) from the Jackson Memorial Laboratory, Bar Harbor, Maine. The quarters were controlled to room temperature and Purina laboratory chow and water were available ad libitum in containers accessible to the dystrophic mice. Mice were weaned at 3–4 weeks with no special transitional diet, and experimental groups formed by dividing litters. A portion of the dystrophic mice were treated with 5 mg MADD in sesame oil per mouse in a single intramuscular dose per week, the dose being equivalent to the methylandrostenediol. Body-weights and mortalities were recorded to the nearest week, and post-mortem examinations were made on mortalities.

Survival time was increased approximately two-fold in the MADD-treated dystrophic mice as compared with untreated controls (Table 1), with no difference apparent between the sexes. Growth was approximately the same in the treated and untreated dystrophic mice with females

Table 1. RESULTS OF MICE TREATED WITH MADD

Group	No. of mice	Median survival-time and range (weeks)	Av. weight at 3 weeks (g)	Av. weight at 13–27 weeks (g)
Normal males	94	not detn.	10	32
Normal females	79	not detn.	9	26
Dystrophic males	13	5 (4–7)	7	dead
Dystrophic females	12	6 (4–9)	6	dead
Dystrophic males receiving MADD	22	13 (7–28)	7	21
Dystrophic females receiving MADD	25	13 (4–27)	7	19