It is well known that, in hypoxia, appetite is initially lost and body weight goes down[15]. The weight loss cannot, however, wholly be explained by the reduction in food intake, because the present high altitude groups lost more weight than the control groups in spite of equal food consumption. An additional mechanism leading to weight loss is dehydration[15]. Plasma volume decreases of up to 26% have been found in rats after at least 10 days of acclimatization to 2400–6100 m[10,16]. Thus the observed increases of blood hemoglobin and hematocrit, changes in the latter running quite parallel to the former, must be partly due to dehydration and partly to a rapid increase of erythropoiesis in hypoxia[15].

The hyperlipidemia of hypoxia in rabbits resembles that caused by repeated bleedings[3,4], all serum lipid classes being elevated. In the present rats, only serum triglycerides showed an increase. This is in contrast to the finding in hemorrhagic hyperlipidemia in rats, in which serum phospholipids and cholesterol increase as well[7]. This increase of serum phospholipids and cholesterol is, however, probably a secondary phenomen to the hypertriglyceridemia. Thus the lack of increase of serum phospholipids and cholesterol in the rats exposed to simulated high altitudes may be explained by the fact that the hypertriglyceridemia was much less marked than that seen in severe bleeding anaemia. The lack of increase of the serum FFA and ketone body concentrations, as well as the slight tendency for triglyceride to accumulate in the liver, are in accordance to findings in hemorrhagic anaemia[7].

It is generally considered that diminished partial pressure of oxygen produces the physiological effects observed at high altitudes[15]. It is interesting to note that cobalt, which is a depressor of cell respiration, also causes marked hyperlipidemia[17]. Thus any type of hypoxia, whether hypoxic, hemic or histotoxic, seems to be connected with hyperlipidemia. Whether this always arises by the same mechanism remains to be studied[18].

Zusammenfassung. Ratten, die 3–12 Tage in einer Luftverdünnung entsprechend einer Höhe von 3000, 5000 und 7000 m gehalten wurden, hatten eine signifikante Hypertriglyceridämie und eine leichte Tendenz zu erhöhten Lebertriglyceridkonzentrationen. Die freien Fettsäuren des Serums waren nicht erhöht.

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Action of Some Compounds on the Adenosine Triphosphatase Activity of *Streptococcus faecalis*

We have previously reported the action of some compounds on the metabolic swelling and glycolytic activity[1] and on the adenosine triphosphate (ATP) pool[2] in protoplasts and/or whole cells of *Streptococcus faecalis*.

The permeability changes responsible for the metabolic swelling seem to be partially dependent of a membrane-bound ATPase[3–5] whose Mg^{++} dependence[6] and sub-unit structure[7] have recently received special attention. In an attempt further to clarify the mechanism of action of those compounds, the present investigation was designed to study their action on the ATPase activity of lysed protoplast suspensions of *S. faecalis*.

Material and methods. S. faecalis ATCC 9790 was grown as reported[1]. The cells were harvested by centrifugation, washed 3 times with redistilled water and twice with 0.075 *M* potassium phosphate pH 6.2, again centrifuged and resuspended in 0.075 *M* potassium phosphate pH 6.2–0.4 *M* sucrose. Muramidase (200 µg/ml) was added, and the mixture incubated at 38 °C in a water bath for 120 min. The resulting protoplasts were harvested by centrifugation, resuspended in a convenient volume of redistilled water to give a protein content between 2.0–2.6 mg/ml, and vigorously stirred. Rapid lysis due to osmotic shock followed. The lysate was diluted 2-fold with $MgCl_2$ 0.01 *M*-*Tris*(hydroxymethyl)aminomethane 0.20 *M*, pH 7.2, and the mixture placed in a water-bath at 38 °C. Additions and sampling were as described before[2], except that glucose was replaced by ATP 2.5×10^{-3} *M*. Following addition of 50 µl of 70% perchloric acid/ml, the samples were assayed for phosphorus by the method of SUMNER[8].

Results and discussion. ATPase activity was an exponential process affected by the compounds tested as represented in the Table. The most pronounced effect was the inhibitory one observed with gramicidin and sodium azide, both of which had an opposite action on the previously studied rate of decay of the ATP in the ATP pool of whole cells metabolizing glucose[2]. Although the 2 processes do not necessarily run parallel since ATPase activity is part but not all of the ATP decay process, it is also important to remember that they were studied in quite different conditions.

Although metabolic differences between whole cells and protoplasts cannot be excluded, it seems now of interest to relate the effect of the tested compounds on the metabolic swelling, glycolytic activity, ATP pool[1,2] and ATPase activity.

The inhibitory action of sodium azide on the metabolic swelling could represent both a slower glycolysis and a

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deviation of the generated ATP from availability to the permeability changes by an accelerated decay process other than the ATPase activity. This last point could also explain, at least in part, the same effect of DNP on the swelling.

From the compounds which increased the rate and degree of reduction in the absorbance of suspensions of protoplasts from *S. faecalis* metabolizing glucose, the action of dicumarol seems now to be related to its capability for increasing energy production. If the suggested increase in the intracellular concentration of K^+ induced by gramicidin[2] proves true, it could account for the same kind of activity of this compound on the swelling. The swelling is known to be stimulated by K^+[3]. In the particular case of arsenate, the disorganization of the internal structure of the cell induced by this compound on *S. faecalis*[9] seems able to explain the more pronounced decrease of the absorbance in swelling protoplasts[10,11].

Influence of various compounds on the linear regression coefficient for Pi liberation from exogenous ATP by lysed protoplast suspensions from *S. faecalis*

	Linear regression coefficient
2,4-Dinitrophenol $1 \times 10^{-3} M$	1.13
Sodium azide $10 \times 10^{-3} M$	0.53
Dicumarol $50 \times 10^{-6} M$	1.06
Gramicidin $22 \times 10^{-6} M$	0.58
Oligomycin $15 \times 10^{-6} M$	0.81
Valinomycin $0.35 \times 10^{-6} M$	0.78
Rutamycin $100 \times 10^{-6} M$	0.92

The value for the control is taken as unity. The linear regression coefficient was determined by the least squares method. An addition of Na-arsenate was followed by a heavy floculation and interference with the phosphorus assay; no results for this compound were recorded.

Résumé. L'activité ATPasique après l'addition d'ATP et en présence de diverses substances est étudié chez *Streptococcus faecalis*. Les effets les plus évidents ont été les effects inhibiteurs de l'acide sodique et de la gramicidine.

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[11] This investigation was supported by grants and fellowships from Fundação Calouste Gulbenkian (to S.M.) and Instituto de Alta Cultura (to C.G.).

Effect of Ecdysone on Glutamic Decarboxylase in Rat Brain

The rapid advances in the understanding of mechanisms of protein synthesis and its genetic control has made the study of the regulation of specific enzyme formation of the greatest current interest. Control of metabolism through induction and repression of the synthesis of specific enzyme proteins is now well established in microbial systems. However, it is now generally agreed that the synthesis of enzyme proteins in animal systems is mediated by hormones. Of special interest in this connection is the insect moulting hormone, ecdysone which has been demonstrated to induce DOPA decarboxylase in *Calliphora*[1,2], a key enzyme in the process of sclerotization. Previous studies[3] indicate that ecdysone may have an effect on mammalian cells also. In the present study, we have investigated the possible effects of ecdysone on the activity of glutamic decarboxylase in rat brain.

Material and methods. Hooded male rats (Long Evans strain) weighing approximately 150 g were used. Ecdysone was solubilized in Tween 20 and doses ranging from 0.25–5 µg/ml were injected i.p. into different batches of rats. The animals were later sacrificed by decapitation at different time intervals of 4, 12 and 24 h. Parallel controls were run under identical experimental conditions (control animals were injected with a solution of Tween 20). For each time interval (including control) 8 animals were sacrificed. Brains were removed immediately and homogenized in $0.02 M$ phosphate buffer (pH 6.4). In the presence of 1-C^{14}-labelled glutamic acid, the homogenate was incubated at 37 °C for 30 min in Warburg manometric flasks. The released radioactive CO_2 was absorbed on hyamine saturated filter paper which was later deposited in scintillation vials containing PPO-POPOP. The radioactivity was counted by means of a liquid scintillation counter (Nuclear Chicago). Further details of the method are essentially the same as described by Lupien and Hinse[4].

Results and discussion. As shown in the Table, there is a significant increase (t-test) in the activity of glutamic decarboxylase in the brains of ecdysone-treated animals as compared to control animals. However, there does not appear to be a well-defined dose-effect relation over the entire range. 24 h after the injection, the increase in enzyme activity is slightly less pronounced compared with its activity at 4 and 12 h after the injection. This could perhaps be due to inactivation or excretion of the hormone. In earlier studies, Burdette and Coda[3] have demonstrated an enhanced rate of mammalian protein synthesis by ecdysone. In our present investigation, whether the increased activity of glutamic decarboxylase represents one of the more general effects of ecdysone

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