variations in the oxygen isotope ratios (*10*).

The results of our measurements are listed in Table 1. For three of the four samples, independent double checks were performed to study the overall reproducibility of the technique. We conclude that measurements of this kind are reliable to within ± 10 percent if a starting sample volume of 500 ml is used. Further reduction of the error limit should be possible.

The few tritium data on Antarctic samples available from the work of Begemann and Libby (*11*) and Shen *et al.* (*12*) show that until 1955 the level was near 17 TU. Our data confirm this general statement concerning the tritium activity at this location prior to nuclear bomb explosions.

Appendix. Dimensioning of the column.

Furry and Jones (*13*) have derived the following expression for the mass transport t [g/sec] of the HT molecules:

$$t = H\bar{c} - (K_c + K_d)\mathrm{d}\bar{c}/\mathrm{d}z \qquad (1)$$

$\bar{c}(z)$ is the average concentration of HT over the horizontal column cross section (number of HT molecules per total number of molecules) and z the coordinate shown in Fig. 2; H, K_c, and K_d are functions of the inner radius r_c of the column, the wire radius r_w, the temperature T_w of the wire, the temperature T_c of the column wall, and the hydrogen-gas pressure. Grove *et al.* (*6*) have obtained experimental values of H, K_c, and K_d, from which the functional dependence on the diameters and temperatures can be evaluated numerically.

When setting the dimensions of the upper (derichment) part of the column, the continuous flow of hydrogen gas must be considered. The net downward HT transport is given by the difference of the downward transport t/ρ [in cm³ of HT molecules/sec; ρ is the density of HT] by thermal diffusion, and the upward transport $V\bar{c}$ resulting from the gas flow (V, flow rate in cm³/sec). In the stationary state one has

$$\frac{t}{\rho} - V\bar{c} = \text{constant}$$
$$\text{for } -L' \leqq z \leqq 0.$$

At $z = -L'$, the total transport must equal $-c_f V$, therefore

$$t = V\rho\,(\bar{c} - c_f). \qquad (2)$$

If Eq. 2 is inserted into Eq. 1 and the differential equation for $\bar{c}$ is solved with the boundary condition $\bar{c} = c_i$ at $z = 0$, the result is

$$\frac{c_i}{c_f} = \frac{H}{H - \rho V} \times \exp\left(\frac{H - \rho V}{K_c + K_d} L'\right) - \frac{V}{H - \rho V}$$

Numerical evaluation of this expression shows that, with $V = 3.6$ liter/hr, the optimal column radius (*14*) is between 1.7 cm at $T_w/T_c = 4$ [T in °K] and 1.9 cm at $T_w/T_c = 2$, and that with $T_w/T_c = 3$, one should obtain $c_i/c_f = 100$ at about $L' = 6$ m. We have chosen $L' = 6.4$ m; $r_w = 1.85$ cm; we expect $c_i/c_f \geqq 100$ for $V < 3.6$ liter/hr and $T_w > 1000$°K.

At $z = 0$ the eigentransport t/ρ of the upper part of the column must equal the eigentransport of the lower part. Therefore the column diameter must not change at this point. This is accomplished by choosing the same diameter in the three "upper" column sections and inserting the sample gas in the middle of the second section (Fig. 1).

The radius of the lowest section has been selected in the following way: According to Eq. 1 the concentration will change with time until $t = 0$, and thereafter

$$\bar{c} = c_i \exp\left(\frac{Hz}{K_c + K_d}\right).$$

For a radius $r_c = 1.35$ cm, the factor $H/(K_c + K_d)$ has its maximum value, almost independently of the temperature ratio. A tube of this radius gives the shortest length L, and such was chosen for the first section.

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Nuclear Mitochondria?

Abstract. *Recognizable mitochondria were detected in the nucleus of a leukemic cell. It is suggested that passage through enlarged nuclear pores, incorporation within a pinched off invagination, or inclusion within the nuclear envelope at telophase may have been responsible for this unusual event.*

During a study concerned with fine-structural aspects of the growth of ascites leukemia cells L 1210 in solid clumps in the abdominal cavity, the presence of a group of structures resembling mitochondria was observed within the nucleus (Fig. 1). One of these bodies (Fig. 2) could be definitely identified, on morphological grounds, as a mitochondrion, in that it showed a double outer membrane and internal cristae. A second body, which could be interpreted as an altered mitochondrion, was surrounded partially by a double membrane and contained what seemed to represent a remnant of cristae. The origin of several other vacuolar structures present in the same area could not be established.

In an attempt to explain such an unusual finding, we can suggest three possible mechanisms whereby these mitochondria may have been "trapped" within the nucleus. It is possible that one or more mitochondria may have "squeezed" through one of the enlarged pores present in the nuclear envelope of these leukemic cells. However, the passage of a particle the size of a mitochondrion through a nuclear pore seems most unlikely. The second possibility is that this unusual picture may represent a transverse section of cytoplasmic structures present in a nuclear invagination; these invaginations are very frequently observed in these cells. However, in all such cases the cytoplasmic structures apparently inside the nucleus appear surrounded by the

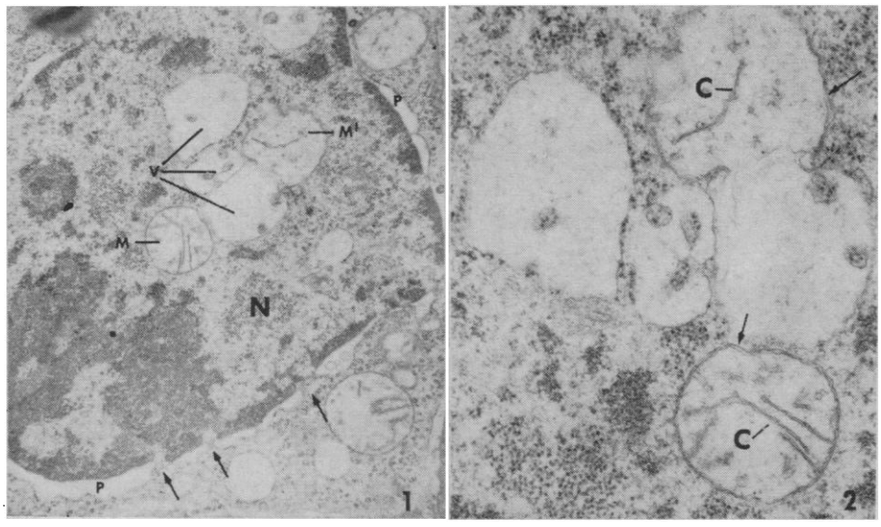

Fig. 1 (left). Portion of L 1210 leukemia cell. The nucleus (*N*) contains a recognizable mitochondrion (*M*), an apparently altered mitochondrion (*M'*), and various undefined vacuolar structures (*V*). The perinuclear cisterna (*P*) shows marked dilations. Prominent nuclear pores are visible (arrows). Glutaraldehyde-osmic acid fixation, Epon embedding, and uranyl acetate counterstaining (× approximately 13,000). Fig. 2 (right). Higher magnification of portion of the nucleus of Fig. 1. Characteristic double membrane (arrows) and cristae (*C*) of the intranuclear mitochondria (*M* and *M'*). Note the absence of nuclear envelope around these structures (× approximately 29,000).

double nuclear membrane. The possibility may then be considered that the "nuclear" mitochondria are derived from such an invagination, and that subsequently the communication between the invaginated area and the general cytoplasm became obliterated and that the portions of nuclear membrane surrounding the trapped mitochondria disappeared. The most likely possibility, however, is to assume that at telophase the mitochondria became trapped within the nuclear envelope as it formed, and this could be considered as an aberration of cell division which may be expected in malignant cells.

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Mercurial-Induced Transformation of Myosin Prevented by Adenosine Triphosphate and Pyrophosphate

Abstract. *Adenosine triphosphate and pyrophosphate prevent the loss of Ca++-activated adenosine triphosphatase activity caused by high concentrations of mercurial sulfhydryl reagent. They concomitantly prevent the transformation of myosin into faster-sedimenting products. This is adduced as support for the hypothesis that the strategic sulfhydryl group is not binding adenosine triphosphate at the active site, but is initiating a conformational change upon its reaction with the mercurial reagent.*

When small amounts of *p*-chloromercuribenzoate are added to myosin, an activation of the Ca++-activated adenosine triphosphatase occurs; when large amounts of *p*-chloromercuribenzoate are added, an inhibition occurs (*1*). However if pyrophosphate or adenosine triphosphate (ATP) are present before the addition of the mercurial sulfhydryl reagent, the inhibition can be reduced or abolished. Morales and Hotta, using pyrophosphate, found that *p*-chloromercuribenzoate in slight excess of the sulfhydryl content of myosin caused no loss of adenosine triphosphatase activity (*2*). Gilmour and Gellert, using ATP, found a low steady rate of adenosine triphosphatase activity even with such a large excess of *p*-chloromercuribenzoate as 122 moles per 10^5 grams of protein (*3*). In a manner similar to its protective action against *p*-chloromercuribenzoate, ATP blocks the reaction of iodoacetamide with the sulfhydryl group whose loss destroys the Ca++-activated adenosine triphosphatase activity (*4*).

Gilmour and Gellert suggest that the sulfhydryl group, whose titration causes inactivation, forms part of the ATP-binding active site (*3*). Stracher also believes that the sulfhydryl is at the active site where it is protected from inactivation by the presence of the substrate (*4*). However Morales and Hotta question how the tremendous affinity of mercury for sulfide could be thwarted by pyrophosphate or ATP actually lodged at the sulfhydryl site (*2*).

The alternative hypothesis to this sulfhydryl group being at the active site is that reaction of this sulfhydryl group in a relatively remote location initiates a conformational change which alters the active site. In the range of mercurial titration where loss of

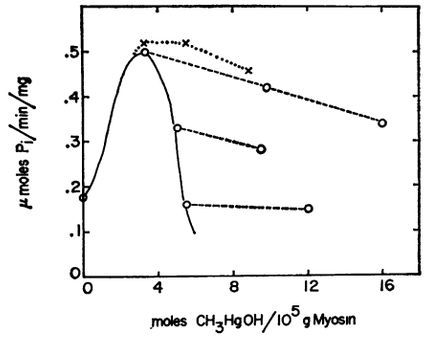

Fig. 1. Effect of methylmercuric hydroxide on Ca++-activated adenosine triphosphatase activity in the presence or absence of ATP or pyrophosphate. Adenosine triphosphatase determinations were performed by *p*H-stat at *p*H 7.0 and 20°C; the zero-order kinetics measured the number of micromoles of base uptake per minute per milligram of protein; colorimetric standardization allowed the data to be expressed as μmoles of inorganic phosphate (P_i). The assay system contained 0.25*M* KCl, 5 m*M* Ca++, and 2 m*M* ATP. Addition of pyrophosphate and methymercuric hydroxide to a sample of myosin was made before addition of 0.3 to 0.5 mg to 4 ml of assay system; addition of methylmercuric hydroxide was also made after the addition. Symbols: open circle, myosin; cross, myosin treated first with pyrophosphate; solid line, methylmercuric hydroxide added in the absence of ATP; dashed line, methylmercuric hydroxide added in the presence of ATP; dotted line, methylmercuric hydroxide added in the presence of pyrophosphate.