In the first set of experiments, to test cytotoxicity, the following mixed lymphocyte cultures were prepared: (C3H + $C3D2F_1$) and (DBA/2 + $C3D2F_1$). The target was a mouse lymphoma line, L5178-Y, originating from DBA/2 ($H\text{-}2^d$) mice and carried both in vivo and in vitro (*11*). Controls consisted of (i) cultures containing cells derived from one donor only, (ii) PHA-stimulated cultures, which were collected on the third day, and (iii) supernatant culture fluid from all of the various cultures. As control for spontaneous ^{51}Cr release, target cells were incubated with fresh medium only; the maximum amount of label that can be released nonspecifically was determined by freezing and thawing a portion of labeled cells three times (Table 1). Only lymphocytes obtained from cultures specifically sensitized against antigens present in the target (C3H + $C3D2F_1$; $H\text{-}2^k$ + $H\text{-}2^{k/d}$) caused a specific release of the label. A significant proportion of the label was already released after 3 hours of incubation and reached 80 to 90 percent of the maximum release at 16 hours (Fig. 2). Cells obtained from the other mixed culture, sensitized against the other set of the hybrid's antigens (DBA/2 + $C3D2F_1$; $H\text{-}2^d + H\text{-}2^{k/d}$), or from PHA-sensitized lymphocytes were ineffective; nor did nonstimulated (nonmixed) cultures or any of the medium controls cause any specific release within the time of observation (Table 1).

To determine the specificity of the target cell, C3H and DBA/2 lymphocytes were similarly sensitized with $C3D2F_1$-hybrid cells in vitro and assayed against lymphoma cell lines originating from either of the parental strains, L5178-Y from DBA/2 and 6C3H-ED from C3H. The release of the ^{51}Cr in these experiments was equally specific and took place only when the sensitization in vitro in the lymphocyte cultures was directed against antigens present in the respective target cells (Table 2). In other words, the sensitized lymphocytes from the combination (DBA/2 + $C3D2F_1$; $H\text{-}2^d + H\text{-}2^{k/d}$) produced release of the label in a significant amount only from 6C3H-ED ($H\text{-}2^k$) cells and not from L5178-Y ($H\text{-}2^d$) cells or vice versa.

Although it has been shown in a xenogeneic system that rat lymphocytes cultivated on mouse fibroblast monolayer do become sensitized against the mouse cells and eventually kill them (*12*), this has not yet occurred in an allogeneic system without the use of a mitogenic agent, such as PHA (*7*). However, a specific sensitization in vitro of mouse lymph node cells by a transplantation antigen preparation results in (i) a specific cytotoxic interaction, as tested by the plaque-reduction assay (*11*), or (ii) in agglutination of sensitized lymphocytes around target cells (*13*). The mixed lymphocyte interaction is considered the initial or "sensitizing" phase of the allograft immune response in vitro. The essential immunologic requirements for the reaction, that is, selectivity and specificity, have also been demonstrated (*2, 9*). Although there was some preliminary evidence that the MLI would culminate in the production of "killer" lymphocytes (*14*), the immunologic specificity of the effector mechanism was not verified.

The destructive phase of the MLI is equally specific. Cytotoxic effect can be demonstrated only in cases where lymphocytes in mixed cultures are sensitized against antigens present in the target. The lack of effect of any of the medium controls, within the time of observation, also rules out nonspecific factors released to the medium (*15*).

In view of the specificity for both the afferent and the efferent responses, it is possible to consider the MLI a complete in vitro model for allograft rejection.

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Mitochondrial RNA Synthesis during Mitosis

Abstract. *HeLa cells arrested in metaphase synthesized relatively normal amounts of mitochondrial RNA, while little RNA synthesis associated with the nucleus was detected. The RNA synthesized resembled the portion of mitochondrial RNA sensitive to ethidium bromide in interphase cells, with major peaks at 21, 12, and 4*S. *Unlike that in interphase cells, RNA synthesis in the mitochondrial fraction of mitotic cells was completely inhibited by ethidium bromide.*

The mitochondria of eukaryotic cells appear to be organelles possessing some degree of autonomy from the nucleus. In addition to DNA, both high molecular weight and 4*S* RNA, which is uniquely associated with the mitochondria of unicellular organisms, have been described (*1*).

The RNA specifically associated with mitochondria has been identified in animal cells. Cytoplasmic RNA which hybridizes with high efficiency to the circular DNA obtained from mitochondria has been described (*2*). Large molecular weight RNA associated with the mitochondria has been further characterized (*3–5*) and consists of two major species plus a large amount of heterogeneous RNA, as judged by acrylamide-gel electrophoresis. These two species have electrophoretic mobilities expected for RNA whose sedimentation constants would be 12 and 21*S* when compared to ribosomal RNA (*4*). The sedimentation velocity of the major species in sucrose-density gradients indicates a slightly different sedimentation constant than that predicted by electrophoretic analysis (*3, 4*). In lieu of a true sedimentation constant determination the values indicated by electrophoresis will

be used here. Low-molecular-weight RNA corresponding to 4*S* is also uniquely associated with mitochondria, and it can be distinguished from bulk cytoplasmic RNA by several criteria (*6*, *7*).

The RNA synthesis associated with the mitochondria can be distinguished from that of the nucleus in several ways. Ethidium bromide is a strongly selective inhibitor of the RNA synthesis system in mitochondria (*5*, *7*). The RNA obtained from mitochondrial fractions of cells grown in tissue culture contains both RNA sensitive to ethidium and a heterogeneous class of RNA resistant to ethidium which appears to be of nuclear origin. The RNA resistant to ethidium may be associated with rough endoplasmic reticulum contaminating these preparations. In addition, nuclear RNA synthesis is much more sensitive than mitochondrial RNA synthesis to inhibition by ultraviolet irradiation (*4*) and virus infection (*8*). The heterogeneous background present in mitochondrial preparations shows the same sensitivity in these agents as does nuclear RNA synthesis.

Here we demonstrate another method of distinguishing nuclear from mitochondrial RNA synthesis. Nuclear RNA synthesis is inhibited in HeLa cells arrested at metaphase, while mitochondrial RNA synthesis continues. Our results are in agreement with the conclusion that RNA sensitive to ethidium is of mitochondrial origin and RNA resistant to ethidium in the mitochondrial fraction originates in the nucleus.

HeLa cells (S-3) were grown in suspension culture as previously described (*9*) and synchronized by the double thymidine block technique (*10*). The synchronization procedure was carried out in suspension culture initially, and after the second release from S phase arrest, the cells were allowed to attach to plastic tissue culture dishes (10-cm dishes, 4×10^6 cells per dish). Colcemid (0.6 μg/ml) was added 5 hours after release from the second thymidine block. About 11 hours after release approximately 90 percent of the cells were accumulated in metaphase. The cells arrested in metaphase, which are loosely attached to the surface (*11*), were pipetted gently from the tissue culture dishes and maintained in suspension culture. The nonmitotic cells, which usually amount to 10 to 20 percent of the population, remained attached to the surface and were not collected. This method yields large

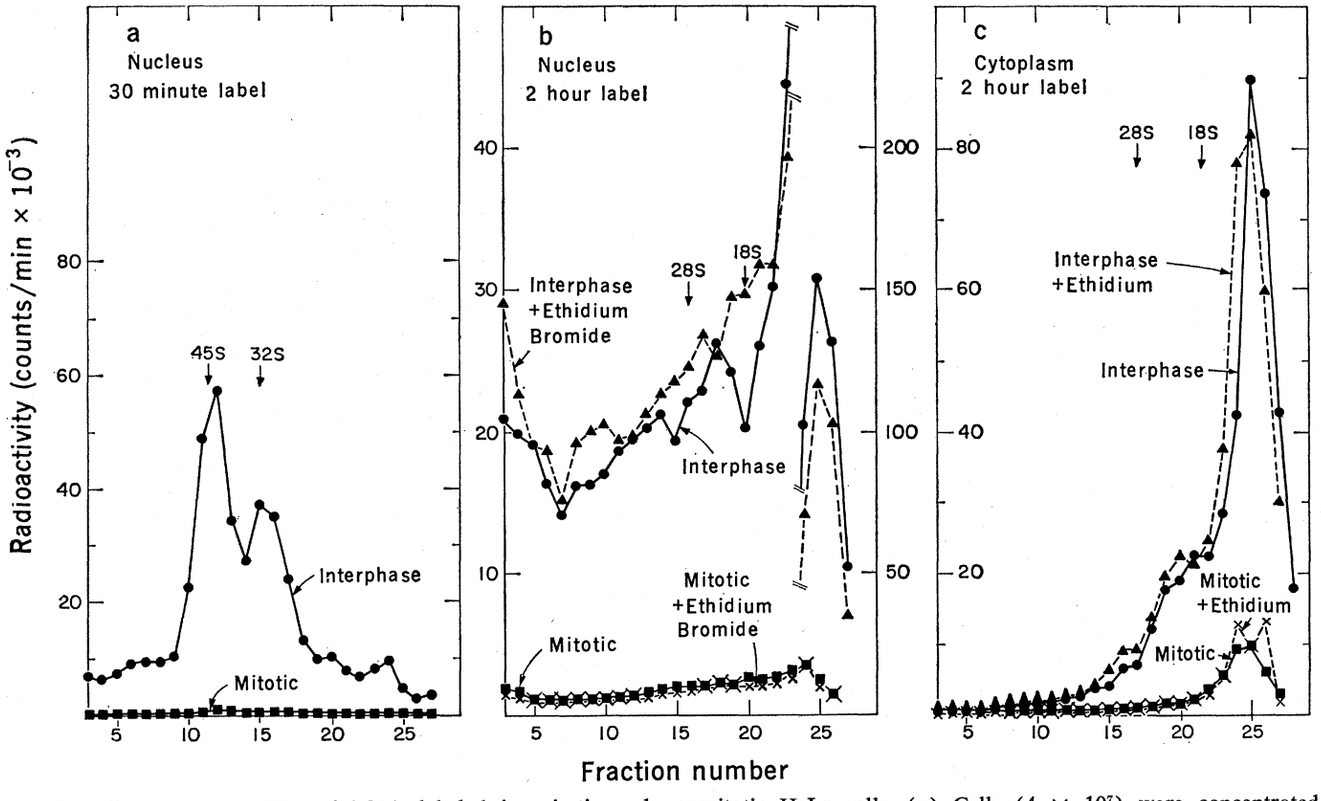

Fig. 1. Sedimentation profiles of RNA labeled in mitotic and nonmitotic HeLa cells. (a) Cells (4×10^7) were concentrated to 2×10^6 per milliliter and 100 μc of [^{3}H]uridine (22 c/mmole was added. After incubation for 30 minutes labeling was stopped by placing the culture tubes in ice. Interphase nuclei or mitotic chromatin was isolated, and RNA was extracted by the hot phenol-SDS method (*12*). The purified RNA was resuspended in 1 ml of SDS buffer and layered onto 15 to 30 percent SDS-sucrose gradients and centrifuged in a Spinco SW25.3 rotor at 25,000 rev/min for 8½ hours. The gradients were fractionated, and the fractions were assayed for radioactivity (*5*). —●—●—, Radioactivity from interphase cells; —■—■—, radioactivity from 97 percent mitotic cells. (b) Cells (4×10^7) were concentrated to 2×10^6 per milliliter and actinomycin D was added to a concentration of 0.04 μg/ml. The cultures were divided in half, and one half was also treated with ethidium bromide at 1 μg/ml. After 30 minutes the cells were labeled as in Fig. 1a, but the period of labeling was 2 hours. The RNA extracted from the nuclear fraction of 93 percent mitotic and interphase cells is shown. Centrifugation was the same as Fig. 1a. —●—●—, RNA from interphase cells; – –▲– –▲– –, RNA from interphase cells treated with ethidium; —■—■—, RNA from mitotic cells; – –X– –X– –, RNA from mitotic cells treated with ethidium. (c) After the mitochondria were removed, aliquots from the supernatants of the cytoplasms from the cells labeled in Fig. 1b were made 0.01*M* in ethylenediaminetetraacetate and 0.5 percent SDS. The samples were layered directly onto 15 to 30 percent SDS sucrose and centrifuged as above. The results were corrected to show radioactivity in the total cytoplasmic supernatant. —●—●—, Radioactivity from interphase cells; – –▲– –▲– –, interphase cells treated with ethidium; —■—■—, mitotic cells; – –X– –X– –, mitotic cells treated with ethidium.

numbers of cells arrested in metaphase with a high degree of purity.

Interphase cells were fractionated by swelling in hypotonic buffer (reticulocyte standard buffer, 0.01*M* NaCl, 0.0015*M* $MgCl_2$, 0.01*M* tris (hydroxymethyl)-aminomethane, *p*H 7.4) followed by gentle homogenization with a Dounce homogenizer (*4*). Immediately after homogenization, sucrose was added to 0.25*M* and nuclei were removed by centrifugation. A crude mitochondrial fraction was prepared by centrifugation of the cytoplasm at 8000 rev/min for 10 minutes. The supernatant was removed, and the mitochondrial fraction was further purified by centrifugation through a sucrose gradient at 25,000 rev/min for 30 minutes (*4*). Mitotic cells were fractionated in the same way. No nuclei are present in mitotic cells; nevertheless the centrifugation deposits over 85 percent of the DNA in the "nuclear" pellet.

The RNA was extracted with hot phenol-SDS (sodium dodecyl sulfate) (*12*) and analyzed either by SDS-sucrose density-gradient centrifugation (*13*) or by acrylamide-gel electrophoresis (*14*).

The major RNA species originating in the nucleus are not labeled during metaphase as shown by autoradiography (*15*). Our data (Fig. 1) confirm these findings by more sensitive measurements of extracted RNA. After the cells were labeled for 30 minutes in [^{3}H]uridine, the total nuclear RNA from interphase and mitotic cells was analyzed in SDS-sucrose gradients (Fig. 1a). The interphase nuclei contained the ribosomal precursor RNA molecules which sediment at 45 and 32*S* as well as a large amount of nuclear heterogeneous RNA (*14*, *16*, *17*). By contrast, the fraction containing the chromatin from mitotic cells showed almost no RNA labeling. The lack of labeled RNA in the chromatin preparation from mitotic cells was not due to loss of RNA to the cytoplasm in the absence of a nuclear envelope because very little labeled RNA was also found in the cytoplasm of these cells.

The nuclei of the cells were incubated for 2 hours with [^{3}H]uridine in the presence of a concentration of actinomycin D (*18*) which selectively suppresses ribosomal RNA synthesis (Fig. 1b) (*17*, *19*). Ethidium bromide, which inhibits mitochondrial RNA synthesis, had little or no effect on nuclear heterogeneous RNA in either interphase or mitotic cells (*5*). The small amount of labeled RNA present in mitotic cell preparations was possibly exaggerated since radioactive precursor may have been exhausted in the interphase culture.

The RNA from the cytoplasms from which the mitochondria were removed yielded the pattern shown in Fig. 1c. A large amount of 4*S* RNA and a small amount of messenger RNA are labeled in interphase cells. The messenger RNA is only partly resolved by the centrifugation used in these experiments. Mitotic cells show a small amount of labeling of transfer RNA (tRNA) which is probably due either to a small interphase contaminant or to labeling of the terminal of preexisting tRNA. Ethidium bromide has no effect on the labeling of these species.

Acrylamide-gel electropherograms of RNA were obtained from the mitochondrial fractions of interphase and mitotic cells (Fig. 2). These are tandem gels with a high concentration of acrylamide at the bottom for displaying the 4*S* RNA and with a low concentration at the top for resolving the higher molecular weight components. The electropherogram of the RNA obtained from interphase mitochondria shows the principal peaks at 4, 12, and 21*S* with the 4*S* RNA partially resolved into several distinct species (Fig. 2a). The 12 and 21*S* RNA's are prominent components of a heterogeneous class of RNA molecules. The RNA labeled in the presence of ethidium bromide constitutes an essentially smooth background which becomes appreciable at the high-molecular-weight end of the gel. There is, in addition, a small peak of RNA resistant to ethidium at 4*S*. This peak originates from polyribo-

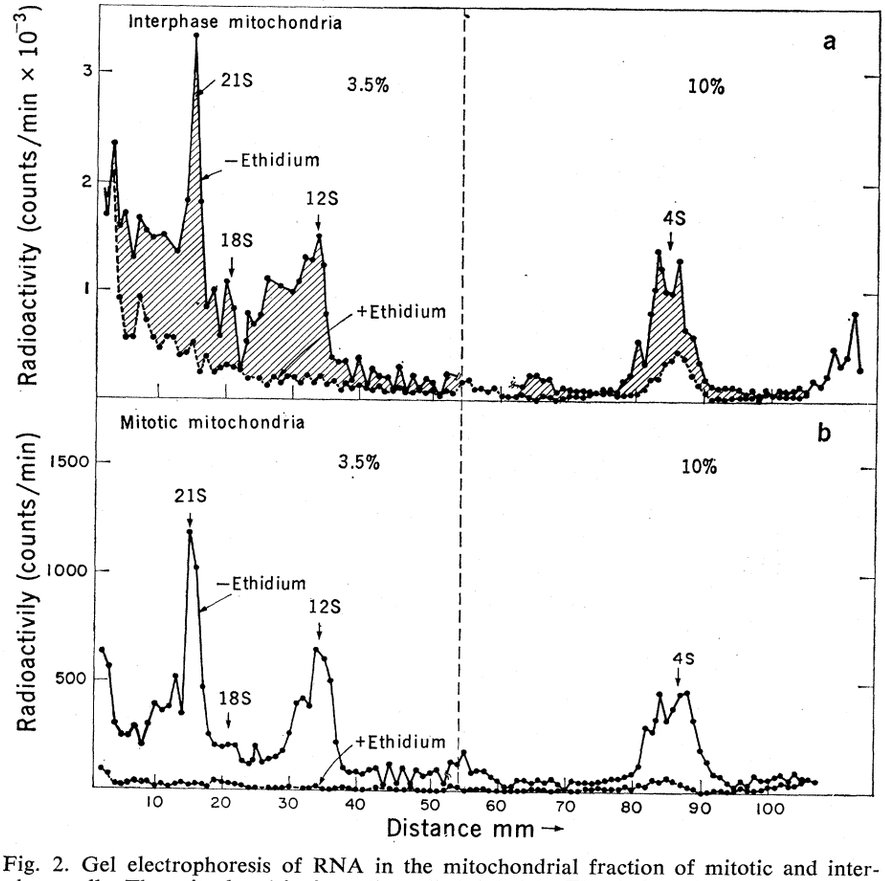

Fig. 2. Gel electrophoresis of RNA in the mitochondrial fraction of mitotic and interphase cells. The mitochondria from the cells labeled in Fig. 1, b and c, were collected by centrifugation at 8000 rev/min for 10 minutes. The crude mitochondrial fraction was then further purified by centrifugation through a sucrose gradient (*3*). The mitochondrial fractions were resuspended in SDS buffer and sonicated to disrupt contaminating DNA. The RNA was then extracted as in Fig. 1, resuspended in 0.1 ml of sample buffer, and layered onto tandem polyacrylamide gels. These gels consist of a 5 cm, 3.5 percent acrylamide gel polymerized on top of a 5 cm, 10 percent acrylamide gel. Electrophoresis was carried out at 10 volt/cm in normal conditions. Gels were fractionated and assayed for radioactivity (*14*). (a) Mitochondrial RNA from interphase cells. —●—●—, Control; – –●– –●– –, treated with ethidium. The ethidium-sensitive material is shown in the shaded area. (b) Mitochondrial RNA from mitotic cells. —●—●—, Control; – –●– –●– –, treated with ethidium.

somes attached to contaminating rough endoplasmic reticulum in these preparations (*5*, *20*).

The labeled RNA obtained from the mitochondria of mitotic cells resembles the RNA sensitive to ethidium of interphase cells (Fig. 2b). There is, however, no RNA resistant to ethidium labeled in the mitochondria of mitotic cells, presumably because RNA synthesis of nuclear origin has been inhibited.

The incorporation of radioactivity in the mitochondrial fraction of mitotic and interphase cells cannot be used to compare the relative amounts of RNA synthesis because the labeling conditions are quite different. The equilibration of the pyrimidine precursor pools with exogenous material is probably quite different in mitotic and interphase cells because in mitotic cells there is no appreciable nuclear RNA synthesis. Also, no attempt was made in these experiments to insure a continuous uptake of exogenous precursor. However, the label in mitochondrial RNA of mitotic cells constitutes a much larger fraction of total cell incorporation than is the case in interphase cells. The radioactivity in the mitochondrial fraction of mitotic cells is therefore not due to incorporation by contaminating interphase cells. Unlike nuclear RNA synthesis, mitochondrial RNA synthesis continues in mitotic cells.

The observation that mitochondrial RNA in mitotic cells is labeled at nearly the same rate as in interphase cells further suggests that radioactive precursors in fact do enter the cell in relatively normal amounts. Thus, the inhibition of radioactive precursor uptake seen at mitosis is due to a true inhibition of RNA synthesis and is not the result of the exclusion of the radioactive isotope.

Two conclusions can be drawn from the results shown in Fig. 2. First, the RNA associated with the mitochondrial fraction and which is sensitive to ethidium bromide is independent of the control mechanisms which inhibit nuclear RNA synthesis at mitosis. Additionally, the complete disappearance of an ethidium-resistant background during the time that nuclear synthesis is inhibited further supports the hypothesis that RNA resistant to ethidium in the mitochondrial fraction is actually of nuclear origin.

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Radio-Frequency Thrombosis of Vascular Malformations with a Transvascular Magnetic Catheter

Abstract. *A tiny magnetic electrode catheter introduced into the human carotid artery has been mechanically and magnetically propelled, with fluoroscopic control, to cranial arteriovenous malformations. Radio-frequency heating of the catheter tip in successive positions occludes abnormal blood vessels.*

Cerebral aneurysms and arteriovenous malformations present a formidable mortality and morbidity in spite of the best existing medical and surgical treatment. All current methods of surgical treatment require an opening in the skull and a transcortical or pericortical approach which in itself may have disadvantages. Indeed some of these conditions cannot be treated by any current technique because of their location or size. We have partly obliterated two extracranial arteriovenous malformations by a new technique that employs a magnetically and mechanically guided intravascular electrode and radio-frequency thrombosis.

The coagulating and hemostatic cutting effects of high-frequency electrical currents have been used in brain surgery since 1926 (*1*) and magnetic guidance of a catheter for selective angiography of the aorta was described in 1951 (*2*). Recently magnetically controlled brain catheters have been used in humans (*3*).

We designed a variety of multipurpose, magnetic-tipped catheters small enough (0.5 mm in diameter) to enter human cerebral arteries. A cylindrical samarium-cobalt magnet (*4*) 0.5 mm in diameter and 1.5 mm in length is

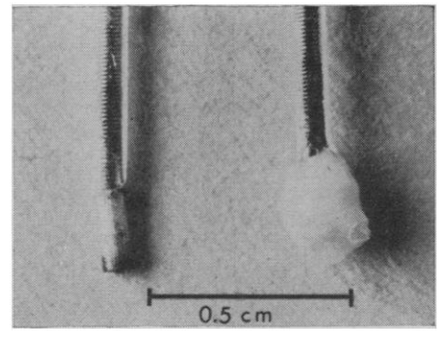

Fig. 1. (Left) Photograph of the transvascular magnetic electrode catheter. (Right) A radio-frequency lesion that has been made by immersing the catheter tip in egg albumin at 70 ma for approximately 5 seconds. The catheter was rapidly moved through the egg white as the lesion was being made to attempt to simulate heat dispersion by blood flow such as might occur in an artery.