A Comparative Study of the Interaction between Concanavalin A and Mitochondria from Normal and Malignant Cells

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SUMMARY

The concanavalin A (con A)-binding capacity of mitochondria isolated from host livers, from Morris hepatomas 7800 and 7777, and from mouse myeloma MOPC-46 were compared. con A binding to the various mitochondrial preparations was quantitated using an isotopic assay procedure in which con A-acetyl-3H is used and by a spectrophotometric agglutination method using native con A. Readily accessible con A-binding sites were determined using washed, intact mitochondria, and total binding sites were evaluated by carrying out incubations in the presence of Triton X-100 (1%). Detergent treatment caused extensive disruption of mitochondrial membranes and increased 2.5- to 3.0-fold the amount of con A-acetyl-3H bound by control liver mitochondria. Tumor-derived mitochondria bound significantly less con A than did the corresponding host liver preparations, indicating an alteration in the glycoproteins of tumor mitochondria. Triton X-100-treated hepatoma 7800 and 7777 mitochondria were capable of binding only 50 and 70% as much con A-acetyl-3H, respectively, when compared with mitochondria isolated from the host liver controls.

The data suggest either that there is a decrease in the concentration of glycoproteins in tumor mitochondria or that there are qualitative changes in the structure of their oligosaccharide side chains. The altered reactivity of tumor mitochondria to con A cannot be accounted for on the basis of differential growth rates of tumor and host liver tissue since mitochondria isolated from regenerating rat liver do not differ from normal rat liver mitochondria in their con A-binding properties.

INTRODUCTION

In recent years, considerable attention has been given to the immunological and biochemical properties of the cell surface of malignant cells with particular emphasis on the complex, carbohydrate-containing constituents of the surface membrane. A variety of experimental approaches have established that, during transformation to the neoplastic state, changes occur in the overall composition and distribution of the glycolipids and glycoproteins of the cell surface (3–5, 7). Useful tools in the study of the surface membrane of neoplastic cells have been the carbohydrate-reactive phytohemagglutinins of which α-mannoside-specific con A is an example. Using tritium-labeled preparations of con A, dramatic changes in the accessibility of agglutinin receptor sites on the cell surface during viral transformation to the oncogenic state have been demonstrated (7).

Much less attention has been devoted to the carbohydrate-containing constituents of subcellular membranes of tumor cells, particularly glycoproteins. Nevertheless, there is evidence that glycoproteins of the endoplasmic reticulum and the mitochondrion are altered in the malignant cell. Wu et al. (17) recently demonstrated pronounced differences in the content and composition of carbohydrates in glycoproteins of mitochondria isolated from normal and SV40 virus-transformed mouse fibroblasts. Both neutral and amino sugar contents were lower in the membranes of organelles of transformed cells.

In this study, mitochondria isolated from host livers and several transplantable tumors have been compared in terms of their con A-binding properties. We demonstrated earlier (11) that con A will bind firmly to purified mitochondria from a variety of mammalian tissues and that the interaction can be inhibited selectively by monosaccharides (e.g., mannose) which specifically bind to this phytohemagglutinin. In the experiments described below, we have isolated mitochondria from transplantable Morris hepatomas 7777 and 7800 and from MOPC-46 mouse myelomas and compared their ability to bind con A with the binding properties of mitochondria derived from the corresponding host livers. We show that there is a significant decrease in the ability of tumor mitochondria to bind con A and that this difference cannot be accounted for by the rapid growth rate of the tumor.

MATERIALS AND METHODS

Tumors. Morris hepatoma-bearing rats (150 to 250 g) were generously provided by Dr. P. Ove of the University of Pittsburgh, Pittsburgh, Pa. Their biology, growth properties, and transplantation procedures are described elsewhere (13, 14). Morris hepatoma 7777 is a rapid growth rate tumor and was used 3 weeks after transplantation.
Morris hepatoma 7800 possesses an intermediate growth rate and was used 4 to 6 weeks after transplantation. The mouse myeloma (MOPC-46) was obtained from Dr. E. C. Heath of the University of Pittsburgh and was used 4 to 6 weeks after transplantation. Although myeloma cells possess no appropriate or readily accessible control tissue, we have compared myeloma-derived mitochondria with those isolated from the host liver. All animals were maintained ad libitum on a regular chow diet and fasted 18 to 24 hr prior to decapitation in order to deplete liver glycogen (6). Partial hepatectomy was performed on 150-g male albino rats under ether anesthesia by removing approximately 70% of the liver.

Preparation of Mitochondria. Animals were killed by a blow to the head, decapitated, and bled for 60 sec. Livers and tumors were rapidly removed and rinsed with ice-cold homogenizing medium of the following composition: D-mannitol, 250 mM; Tris buffer, pH 7.4, 50 mM; and EDTA, 1 mM. The tumors were trimmed of necrotic, nontumorous material. The livers and tumors were minced with scissors, suspended in a Potter-Elvehjem tissue grinder (50 ml capacity) and homogenized (3 volumes of homogenizing medium per g of tissue) with 5 passes of a motor-driven, radially serrated, rotating Teflon pestle. The homogenate was diluted with homogenizing medium to a final volume 10 times that of the original weight of tissue and filtered through 4 layers of cheesecloth. Centrifugation was carried out at 800 × g for 20 min. Unless otherwise stated, all procedures were carried out at 2–4°. The supernatant was passed through 2 layers of tissue paper to remove excess lipid and centrifuged for an additional 5 min at 800 × g. The supernatant was centrifuged for 10 min at 6,800 × g and the fluffy layer was discarded. The pellet was washed twice by resuspension in one-third the original volume of homogenizing medium followed by centrifugation at 6,800 × g for 10 min. The washed mitochondrial pellet was resuspended in homogenizing medium and layered onto a continuous, linear sucrose density gradient prepared by mixing 20% w/w and 54% w/w sucrose solutions in Beckman cellulose nitrate tubes (1 x 3 inches). After centrifugation for 3 hr at 63,500 × g, approximately 20 fractions were collected by puncturing the bottom of the tube with an 18-gauge needle. The refractive index of each fraction was determined using an Abbe refractometer. Based on quantitative enzyme marker analysis, mitochondria prepared by this procedure have been shown (11) to be considerably more pure than those prepared using only differential centrifugation methods.

Protein Determinations. Mitochondrial protein was determined by the method of Lowry et al. (12) using human serum albumin (Fraction V, Sigma Chemical Co., St. Louis, Mo.) as standard. Concentrations of con A were determined by absorption at 280 nm assuming an E1%1 cm of 11.4 (2).

Preparation of con A. con A was prepared by the method of Agrawal and Goldstein (1). Tritium-labeled con A was prepared by acetylation of the native protein with 3H-labeled acetic anhydride (specific activity, 50 mCi per nmole) by the method of Fraenkel-Conrat (9). Acetylation did not significantly alter the ability of con A to precipitate glycogen from solution.

con A-binding Determinations. The binding of con A to mitochondria was quantitated by 2 procedures, one using a spectrophotometric determination of the amount of turbidity developed when Triton X-100-treated preparations of mitochondria were incubated with native con A and an isotopic binding method using the tritium-labeled agglutinin. The spectrophotometric method requires the presence of a detergent such as Triton X-100 during the assay since intact mitochondria are extensively agglutinated by con A and settle rapidly from solution thus making quantitative determinations difficult. The inclusion of detergent permits the formation of a stable, turbid complex between mitochondria and con A which can be reproducibly quantitated by absorbance measurements. For each of the binding assays, mitochondria were isolated from continuous sucrose density gradients (10,000 × g for 10 min) and washed twice with a standard buffer containing 100 mM potassium phosphate, pH 7.0, and 1% sodium chloride. Under these conditions, there was no extraction or release of con A-reactive material into the supernatant and all binding activity was recovered in the mitochondrial pellet. In the spectrophotometric assay, incubations contained the following components in a final volume of 1.0 ml: potassium phosphate, 100 mM, pH 7.0; sodium chloride, 1%; Triton X-100, 1%; 0 to 0.8 mg of mitochondrial protein; and 2 mg of native con A. The reaction was initiated by the addition of con A and the absorbance at 500 nm was monitored continuously at 22° using a Gilford recording spectrophotometer. The omission of either con A or mitochondrial protein from the incubation mixture resulted in absorbance changes which were less than 5% of that observed in the complete incubation. Maximum absorbance changes were usually obtained within 50 to 60 min (11). Binding data are expressed in terms of the maximum absorbance change produced per mg of mitochondrial protein, and all binding data reported were obtained from at least 3 determinations using various amounts of mitochondrial protein (0.1 to 1.0 mg). We previously demonstrated (11) that the interaction between mitochondrial protein and con A is inhibited by greater than 95% by methyl-α-D-mannopyranoside and less than 15% by α-galactose when these monosaccharides are present in the incubation at final concentration of 10 mM.

The isotopic binding assay using con A-acetyl-3H involves vacuum filtration of incubation mixtures through glass fiber filters. Incubation mixtures contained the following components in a final volume of 0.06 ml: potassium phosphate buffer, 5 μmoles, pH 7.0; sodium chloride, 0.5 mg; con A-acetyl-3H, 0.01 mg (1 × 10⁶ cpm/mg); 0 to 0.2 mg of mitochondrial protein; and Triton X-100 (1%) as indicated. Incubations were carried out at 22° and after 50 min were diluted to 2.0 ml with the standard buffer described above and vacuum-filtered rapidly through Whatman glass fiber filters (2.4 cm, Grade GF/C). The filters were washed with 20 ml of the same buffer and counted in 10 ml of a Triton X-100 toluene scintillation fluid (10).

Enzyme Assays. Cytochrome oxidase was assayed at 22° using dithionite-reduced equine cytochrome c (Sigma), according to the procedure of Cooperstein and Lazarow (8). The assay was linear with time and amount of mitochon-
drial protein, and 1 unit of activity is defined as the amount of enzyme that catalyzes the formation of 1 nmole of product per min.

RESULTS

Analysis of Tumor and Host Liver Mitochondria by Sucrose Density Gradient Centrifugation. In order to obtain relatively pure preparations for con A-binding studies, mitochondria from tumors and host livers were first isolated by differential centrifugation and then subjected to centrifugation in linear gradients of sucrose. The results of analysis of gradients yielded profiles similar to those reported previously (11). In all cases cytochrome oxidase activity was coincident with protein concentration in the region of the gradients containing mitochondria. Considering the 3 tumors studied, no consistent difference was observed in the isopycnic banding position of tumor mitochondria relative to host liver mitochondria (Table 1). Mitochondria isolated from hepatoma 7800 appeared to be less dense than corresponding host liver mitochondria whereas hepatoma 7777-derived mitochondria were slightly more dense than those isolated from the host liver. In the case of MOPC-46 myeloma, the tumor-derived mitochondria were considerably less dense than those isolated from the host liver. Results of cytochrome oxidase assays on these mitochondrial preparations are also summarized in Table 1. In general, the cytochrome oxidase specific activity of tumor mitochondria was slightly greater than that of mitochondria obtained from the corresponding host livers. This observation has also been reported for a number of Morris hepatomas with a variety of growth rates (15, 16).

The Quantitative Analysis of the Binding of con A to Tumor and Host Liver Mitochondria. The more sensitive method for quantitatively evaluating the binding of con A to various mitochondrial preparations is the isotopic assay using con A-acetyl-^3H. An additional advantage of this isotopic binding assay when compared to the spectrophotometric agglutination assay is that the former is capable of measuring available or accessible binding sites on intact, washed mitochondria as well as the total number of binding sites made available by the addition of detergent to the assay medium. In order to evaluate the reproducibility of the binding assay, the livers from 3 fasted rats were removed and mitochondria were isolated separately from each. The ability of each mitochondrial preparation to bind con A-acetyl-^3H in the presence and absence of Triton X-100 was compared, and the results of this study are summarized in Table 2. The con A-binding capacity of the 3 preparations of mitochondria, expressed on a per mg protein basis, is reproducible and differs from the mean by less than 10%. The addition of Triton X-100 to each of the mitochondrial preparations caused a marked increase (approximately 2.5-fold) in the amount of con A-acetyl-^3H which was bound.

The stimulation of con A binding to mitochondria by Triton X-100 is probably due to disruption of membranes and unmasking of otherwise buried binding sites rather than to an increase in the intrinsic affinity of con A for particular carbohydrate residues since we have observed that the concentration of detergent used in these studies has no effect on the precipitation of glycogen by con A-acetyl-^3H. Therefore, these results indicate that the isotopic binding assay is a reliable and reproducible method for comparing the relative number of con A-binding sites present in different mitochondrial preparations.

The reactivity of mitochondria isolated from the Morris hepatoma 7800 and host liver is compared in Chart 1. The amount of con A-acetyl-^3H bound is linearly proportional to mitochondrial protein concentration, and from the slope of these data the amount of labeled con A bound can be expressed on a per mg mitochondrial protein basis. In the absence of detergent, washed, intact host liver mitochondria bind significantly more con A-acetyl-^3H than the tumor-derived preparation. When compared in the presence of Triton X-100, tumor mitochondria bind only one-half as much con A as the corresponding host liver preparation.

The decreased content of con A-reactive constituents in hepatoma 7800 mitochondria relative to the control preparation was confirmed using the spectrophotometric agglutination assay procedure. As shown in Chart 2, the addition of con A to detergent-treated mitochondria resulted in agglutination, which produced maximum absorbance changes in approximately 50 min. Since the absorbance change produced by con A addition is linearly proportional to mitochondrial protein concentration (11) binding data obtained by this method can be expressed on a per mg mitochondrial protein basis. Data presented in Chart 2 show that the con A-binding capacity of hepatoma 7800 mitochondria is approximately one-half that of host liver mitochondria. This observation agrees with results obtained using the isotopic binding procedure described above. Table 3 presents a summary of the results of binding experiments in which the relative reactivity of tumor and host liver mitochondria to con A were compared. The con A-binding capacity of tumor-derived mitochondria was significantly lower than the host liver control preparations for all tumors studied. According to the turbidometric spectrophotometric assay carried out in the presence of Triton X-100, mitochondria from hepatomas 7800 and 7777 were 50 and 30% less reactive than

<table>
<thead>
<tr>
<th>Source of mitochondria</th>
<th>Buoyant density* (g/ml)</th>
<th>Cytochrome oxidase (units/mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatoma 7800</td>
<td>1.175 ± 0.003*</td>
<td>510</td>
</tr>
<tr>
<td>Host liver</td>
<td>1.185 ± 0.003</td>
<td>398</td>
</tr>
<tr>
<td>Hepatoma 7777</td>
<td>1.200 ± 0.004</td>
<td>440</td>
</tr>
<tr>
<td>Host liver</td>
<td>1.196 ± 0.003</td>
<td>285</td>
</tr>
<tr>
<td>MOPC-46 myeloma</td>
<td>1.175 ± 0.003</td>
<td>358</td>
</tr>
<tr>
<td>Host liver</td>
<td>1.190 ± 0.004</td>
<td>311</td>
</tr>
</tbody>
</table>

* Determined from the equilibrium position of mitochondria on linear continuous sucrose density gradients.

* Mean ± S.D. on 3 to 5 tissues.
Table 2

Analysis of the reproducibility of the binding of con A-acetyl-\(^{14}\)H to rat liver mitochondria

Liver mitochondria were isolated separately from 3 animals by differential centrifugation and sucrose density gradient procedures described in "Materials and Methods." Mitochondria were washed with standard potassium phosphate-sodium chloride buffer and then analyzed for their capacity to bind con A-acetyl-\(^{14}\)H in the presence (+) and absence (−) of Triton X-100 (1% final concentration).

<table>
<thead>
<tr>
<th>Animal</th>
<th>Triton X-100 (1%)</th>
<th>Triton X-100 (1%)</th>
<th>(Specific activity + Triton X-100)/ (specific activity − Triton X-100)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>1.00</td>
<td>0.415</td>
<td>2.4</td>
</tr>
<tr>
<td>2</td>
<td>1.07</td>
<td>0.377</td>
<td>2.8</td>
</tr>
<tr>
<td>3</td>
<td>0.95</td>
<td>0.372</td>
<td>2.5</td>
</tr>
<tr>
<td>Mean</td>
<td>1.01</td>
<td>0.388</td>
<td>2.6</td>
</tr>
</tbody>
</table>

Chart 1. Quantitative analysis of the binding of con A-acetyl-\(^{14}\)H to mitochondria from Morris hepatoma 7800 and host liver. Mitochondria were isolated from Morris hepatoma 7800 and host liver by differential centrifugation and sucrose density gradient methods and were washed with standard potassium phosphate-sodium chloride buffer according to "Materials and Methods." The amount of con A-acetyl-\(^{14}\)H bound to various amounts of tumor (x) and host liver (O) mitochondria was determined in the presence (a) and absence (b) of Triton X-100 using the isotopic assay described in "Materials and Methods."

host liver mitochondria and, in the case of the myeloma (MOPC-46), tumor-derived mitochondria were only one-half as reactive as those obtained from the host liver. These observations were supported by the results obtained using the isotopic binding assay carried out in the presence of Triton X-100; in all cases, tumor-derived mitochondria bound considerably less con A-acetyl-\(^{14}\)H than host liver mitochondria.

Since Triton X-100 causes extensive disruption of mitochondrial membranes, data derived from binding studies...
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Conducted in the presence of this detergent probably reflect the total number of con A-reactive sites in the preparation. Using con A-acetyl-\(^3\)H it is therefore possible to distinguish between the total number of con A-binding sites and the number of accessible sites by conducting assays in the presence or absence of Triton X-100, respectively. A means of expressing the relative accessibility of con A-reactive sites is the ratio of the specific activity (cpm bound per mg mitochondrial protein) of con A-acetyl-\(^3\)H bound in the presence and absence of detergent. In the case of liver mitochondria from normal, non-tumor-bearing rats, this ratio is approximately 2.5 (Table 1), indicating that only about 40% of the total con A-reactive sites are exposed or accessible to the labeled phytohemagglutinin. Similarly, with respect to mitochondria from the host liver of hepatoma 7777-bearing animals, the ratio of the specific activity of con A-acetyl-\(^3\)H bound in the presence of Triton X-100 to that in the absence of Triton X-100 was 2.7, indicating that the majority of the con A-binding sites were inaccessible to the labeled phytohemagglutinin. In contrast, the binding of con A-acetyl-\(^3\)H to mitochondria from hepatoma of the same animal was stimulated only 40% by the addition of Triton X-100, indicating that in the tumor mitochondria a relatively large percentage of the total con A-binding sites are accessible to the tritium-labeled protein.

In summary, these results indicate that tumor mitochondria are significantly reduced in their content of con A-reactive constituents and that these deficient components are probably missing from a compartment of the mitochondrion which is inaccessible to con A-acetyl-\(^3\)H.

Analysis of the con A Reactivity of Mitochondria Isolated from Regenerating Rat Liver after Partial Hepatectomy. In order to determine whether the decreased reactivity of tumor mitochondria to con A could be attributed to the relatively rapid growth rate of the tumor as compared to that of the host liver, mitochondria from rapidly growing, regenerating rat liver were tested for their ability to bind con A. Mitochondria were isolated from the livers of rats on the 2nd, 4th, and 7th days after partial hepatectomy, and their ability to bind con A-acetyl-\(^3\)H and native con A was evaluated in the presence of Triton X-100. The results presented in Table 4 demonstrate that there is no significant difference between the con A reactivity of mitochondria isolated from the liver of a control animal and those isolated from liver tissue undergoing rapid cell division at various periods during regeneration. We conclude that the decreased reactivity of tumor mitochondria to con A cannot be attributed simply to the rapid growth rate of the tumor.

DISCUSSION

The results of the experiments presented here demonstrate that con A will react with purified mitochondria from several tumors and from normal and regenerating rat liver. We have shown previously (11) that the interaction between con A and mitochondria can be inhibited specifically by methyl-\(\alpha\)-D-mannopyranoside. The extraction of lipids did not reduce the agglutinability of mitochondria by con A, and trypsinization and periodate oxidation of soluble mitochondrial proteins substantially decreased their ability to bind con A. These observations are consistent with the belief that con A reacts primarily with glycoprotein components of the mitochondrion. In addition, at least 5 soluble, periodic acid-Schiff base-staining, high-molecular-weight components from rat liver mitochondria can be resolved by disc gel electrophoresis. Assuming that glycoproteins are the major con A-reactive species associated with mitochondrial preparations, our quantitative binding data concerning tumor and host liver mitochondria therefore reflect quantitative and/or qualitative alterations in the glycoproteins of tumor mitochondria.

Several explanations could account for the decreased con A-binding capacity of tumor mitochondria. The decreased reactivity of tumor mitochondria could simply reflect a decrease in the overall concentration of all species of mitochondrial glycoprotein in the absence of any change in either the number of specific glycoproteins or the structure

<table>
<thead>
<tr>
<th>Source of mitochondria</th>
<th>Spectrophotometric assay ((\Delta A_{460}/\text{mg protein}))</th>
<th>con A-acetyl-(^3)H assay (cpm bound/mg protein x (10^{-4})) + Triton X-100</th>
<th>(Specific activity + Triton X-100) - (specific activity - Triton X-100)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatoma 7800</td>
<td>0.49 (50)*</td>
<td>0.78 (64)</td>
<td>2.24 (54)</td>
</tr>
<tr>
<td>Host liver</td>
<td>0.98</td>
<td>1.23</td>
<td>4.15</td>
</tr>
<tr>
<td>Hepatoma 7777</td>
<td>0.46 (70)</td>
<td>1.69 (113)</td>
<td>2.40 (61)</td>
</tr>
<tr>
<td>Host liver</td>
<td>0.66</td>
<td>1.50</td>
<td>3.98</td>
</tr>
<tr>
<td>MOPC-46 myeloma</td>
<td>0.46 (49)</td>
<td>0.37 (100)</td>
<td>0.98 (64)</td>
</tr>
<tr>
<td>Host liver</td>
<td>0.94</td>
<td>0.37</td>
<td>1.53</td>
</tr>
</tbody>
</table>

* Assay mixtures contained Triton X-100 (1%).
* The number in parentheses indicates the specific activity of the con A binding of tumor mitochondria as a percentage of control (host liver) mitochondria.
of the carbohydrate side chains in these glycoproteins. This result would be obtained if in the tumor cell there were a decrease in the activity of pathways of sugar metabolism leading to the transfer of sugar residues to mitochondrial acceptors. Wu et al. (17) have discussed possible alterations in carbohydrate metabolism and glycosyl transferase enzymes that could account for changes in the concentration and structure of membrane glycoproteins observed during virus transformation. Since mannose is a major neutral sugar component of mitochondrial membranes (17) it may be that the decreased reactivity of tumor mitochondria is due to a decrease in the number of available α-mannosyl residues resulting from: (a) the incomplete transfer of mannose residues to mitochondrial acceptors or (b) to the masking of mannose residues by other monosaccharides.

In an earlier communication (11), we reported that the majority (70%) of the con A-reactive components of the mitochondrial could be readily solubilized during a digitonin submitchondrial fractionation procedure and that this major fraction of con A-reactive material cofractionated with adenylate kinase, marker for the intracristate space. Because of this fact and since the tumor mitochondria that we have studied are significantly reduced (30 to 50%) in their complement of con A-reactive constituents relative to host liver preparations, it is likely that alterations will be found in the glycoproteins of the adenylate kinase-rich, intracristate space fraction of tumor mitochondria.

In considering available or accessible con A-binding sites versus buried or inaccessible sites, in general it appears that the fraction of the total number of lectin-binding sites that are readily accessible to phytohemagglutinin (determined in the absence of Triton X-100) in normal host liver mitochondria is less than in tumor-derived mitochondria. For example, the inclusion of Triton X-100 in binding assays involving hepatoma 7777 mitochondria stimulated the amount of con A-acetyl-3H bound by only 40%, whereas the binding of labeled con A to liver mitochondria from the same animal was stimulated nearly 3-fold (Table 3).

Several observations suggest that the decreased reactivity of tumor mitochondria to con A cannot be attributed to differences in growth rate of tumors and host livers. First, mitochondria isolated from regenerating livers with rapid growth rates were indistinguishable from normal liver mitochondria when subjected to the quantitative con A-binding studies described above (Table 4). Secondly, there was no correlation between the growth rate of the 2 hepatomas studied and the extent to which mitochondria isolated from these tumors exhibited differences in con A-binding properties relative to the respective host liver control preparations. In fact, the intermediate growth rate hepatoma 7800 yielded mitochondria with less con A-binding capacity relative to host liver mitochondria than did the rapid-growth rate hepatoma 7777. Studies involving the subfractionation of mitochondrial compartments and the resolution of specific mitochondrial glycoproteins are required in order to determine the basis for the altered reactivity of tumor mitochondria to con A.

REFERENCES

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