ABSTRACT

We have determined rates of fatty acid (FA) synthesis from glucose carbon and all two-carbon units in control mice and in mice bearing Ehrlich ascites carcinomas. Using \( [U-^{14}C] \)glucose and \( ^3 \)H\(_2\)O as tracers under three nutritional conditions (24-hr fasted, 24-hr fasted-refed, and \textit{ad libitum} fed-refed), we found that lipogenic regulatory mechanisms in adipose tissue and livers of mice bearing advanced tumors were similar to those of control mice. FA synthesis from glucose carbon and from all two-carbon units in livers of tumorous (8-day tumors) mice was at least as fast as that in control mice in the fasted and fasted two-carbon units in livers of tumorous (8-day tumors) mice was at least as fast as that in control mice in the fasted and fasted-refed states but only about one-half that of controls in the fed-refed condition. The rate of FA synthesis from two-carbon units in carcasses of mice with 8-day tumors was not significantly different from that of controls in any of the 3 dietary states studied; however, in fed-refed mice with 8-day tumors, the rate of FA synthesis in the whole body was only one-half that of controls. The rate of FA synthesis from glucose carbon in carcasses of these tumorous mice was significantly depressed compared to that of controls in both the 24-hr-fasted and the fed-refed states. In well-nourished mice with early (5-day) tumors, the whole-body lipogenic rate from all two-carbon units was not depressed. Thus, decreased lipogenesis observed in host tissues of mice with advanced tumors is due to malnutrition; this secondarily depressed lipogenic activity probably contributes significantly to the loss of body fat that may occur at later stages of tumor growth. De novo FA synthesis in Ehrlich ascites cells, although small compared to that of the whole-body rate, was substantial in relation to lipids needed for tumor nutrition.

INTRODUCTION

We have been interested in 2 aspects of lipid metabolism in cancerous animals. One aspect deals with the effects of cancer growth on fat metabolism in the host; the other deals with the dependency of the cancer upon the host for its supply of FA\(^3\) for growth and energy needs. Although earlier workers (17, 19–21) have studied these questions, they did not use quantitative techniques that would allow meaningful calculations of FA-synthetic rates both in the host and cancer tissues under different dietary conditions. Moreover, none of the earlier studies were done under conditions that would ensure maximal lipogenic activity (5). A tumor-induced defect would be most readily detected when the important regulatory processes in lipogenesis are operative, \( i.e. \), during the actual absorption of carbohydrate when lipogenesis from glucose carbon may be 100 to 1000 times faster than it is in the fasted state (8).

In the present study, we have measured rates of \textit{de novo} FA synthesis \textit{in vivo} from glucose carbon and from 2-carbon units in host tissues and in a common experimental cancer, the Ehrlich ascites carcinoma. These studies were carried out at 2 stages of tumor growth: early (5 days), when the tumor was barely visible and food intake was normal; and later (8 days), when food intake was reduced. These studies have allowed us to reevaluate the relative roles of lipogenesis and plasma FFA transport to the tumor from the host's circulation and to determine whether a tumor-induced inhibition of FA synthesis occurs in the host's tissues.

MATERIALS AND METHODS

Experimental Protocol

Two studies were carried out using male Swiss-Webster mice (Hilltop Laboratory Animals, Inc., Chatsworth, Calif.). One-half of the animals were inoculated with a subline of Ehrlich ascites carcinoma (15 x 10\(^6\) cells/mouse) originally obtained from Dr. R. W. McKee, UCLA School of Medicine.

\textbf{Study 1.} In this study, we determined lipogenic rates in mice bearing 5-day-old tumors under the following 3 dietary conditions: fasted 24 hr; fasted 24 hr and refed a test meal of 50% glucose as an aqueous solution; and fed \textit{ad libitum} and intubated with a 50% aqueous solution of glucose (fed-refed). Mice were divided into control and tumor groups 5 days prior to tumor inoculation. They were maintained on a 58% glucose (fat-free) diet (5) and were given water \textit{ad libitum}. Body weights (g) of mice on the day of the experiment were as follows: 24-hr-fasted group, controls, 31.2 ± 0.9 (S.E.) \( (n = 15), \) tumor-bearing, 41.5 ± 1.1 (\( n = 15), \) \textit{ad libitum}-fed groups, controls, 33.8 ± 0.8 (\( n = 5), \) tumor-bearing, 47.1 ± 0.8 (\( n = 6). \) The average tumor volume was 4.1 ± 0.3 ml (\( n = 21). \)

\textbf{Study 2.} In the second study, we determined lipogenic rates in host tissues at an early stage of tumor development, namely, in mice bearing 5-day-old tumors. The control and tumor groups were placed on the fat-free diet 2 days before tumor inoculation. Rates of FA synthesis were determined in control and in tumor-bearing mice on the experimental day (Day 5) in the fed-refed state only. Body weights of controls and tumor groups on Day 5 were as follows: controls, 32.5 ± 0.8 g (\( n = 14), \) tumor-bearing, 37.9 ± 1.1 g (\( n = 14). \) Increases in body weights over Day 0 values for control and tumor groups were: controls, 1.9 g; and tumor bearing, 3.8 g. The food intake of tumor-bearing mice was not significantly different from that of control mice.
Calculation of Rates of FA Synthesis from all 2-carbon sources using [U-14C]glucose and 3H2O, that we and others have developed over a number of years to see and others have developed over a number of years to

**Injection and Sampling**

In the first study, [U-14C]glucose was injected i.v. into 24-hr-fasted control mice and mice bearing 8-day-old tumors in order to measure rates of FA synthesis (see Calculation of Rates of FA Synthesis). Heparinized microcapillaries were used to collect blood from the ophthalmic sinus (9) at 1, 5, 15, 30, and 60 min after injection. The blood samples were immediately spun, and the plasma was used to determine plasma glucose specific activity (7).

**Radioactive Tracer Doses**

In the first and second studies, 3H2O (1 to 2 mCi; 50 μl/mouse) was injected s.c. A 50% aqueous solution of [U-14C]glucose (5 μCi; 0.5 ml) was administered by gastric intubation. Control and tumor-bearing mice that were either fasted for 24 hr or fed ad libitum were refed (120 mg of labeled glucose per 20 g of mouse) i.g. 15 min after 3H2O injection. All mice were killed 60 min after glucose feeding (75 min after 3H2O injection).

**Analyses**

Carcasses and livers were saponified in 30% (50% ethanolic) KOH, 110 and 10 ml, respectively, for 2 to 3 hr under reflux in an 80°C water bath. FA extraction and radioactivity assay procedures were the same as described earlier (8).

Plasma glucose specific activity was determined using long thin-layer chromatogram strips (7) as described previously. Plasma glucose concentration was determined by enzymatic assay of deproteinized samples of blood plasma (2, 7).

Body (plasma) 3H2O specific activity was determined by radioassay of diluted plasma and by weighing aliquots of plasma before and after drying in air. Negligible counts remained in plasma solids after evaporation.

Standard techniques were used for the radioassay of doubly labeled samples. Appropriate standards were assayed simultaneously, and quench corrections were applied when necessary.

**Calculation of Rates of FA Synthesis**

The approach used is a combination of several techniques that we and others have developed over a number of years to study in vivo rates of FA synthesis from glucose carbon and from all 2-carbon sources using [U-14C]glucose and 3H2O, respectively, as tracers (8, 22). Calculations of rates are based upon the use of simplified models and assumptions that have been stated in our earlier reports (4, 8, 22).

**Rates of FA Synthesis from all 2-Carbon Units Using 3H2O**

<table>
<thead>
<tr>
<th>nmol FA synthesized/min</th>
<th>= (3H cpm in TLFA in t (min)</th>
<th>3H cpm/g atom H in body water</th>
<th>X 10^6</th>
<th>X 1</th>
<th>13.3</th>
<th>(t2 - t1)</th>
</tr>
</thead>
</table>

We carried out separate experiments to confirm that near-equilibrium between plasma and total body 3H2O was reached rapidly enough to allow the above calculation. A plateau value corresponding to a body water space of 70 ± 1.4% of body weight (n = 10) was reached within 10 min after i.p. injection. Calculations were carried out to obtain minimal estimates of lipogenic rates in the case of fasted-refed mice that had received injections of 3H2O at t₀, had been fed glucose at t = 15 min, and had been killed at t = 75 min. Although the lipogenic rate is not linear in these mice (8), the data were treated as though it was. By using a 75-min interval, we have underestimated the true maximal rate by about 10% (8).

**Rates of FA Synthesis from Glucose Carbon.** Our theoretical approach was the same as that described and used in our previously published studies of lipogenic activation in mice (5, 6, 8). Briefly, the approach was as follows. Some mice were fasted for 24 hr and then given i.v. injections of tracer [U-14C]glucose. The endogenous flux of glucose carbon to TLFA in tissues of fasting mice was determined using semicompartamental analysis of plasma glucose specific activity data, in this case collected during a 60-min interval, and of 14C incorporated into TLFA at 60 min after tracer injection (4, 8). In fasted-refed or fed-refed mice, instead of injecting labeled glucose, we fed the animals a 50% aqueous [U-14C]glucose test meal (120 mg/20 g of body weight) and measured the incorporation of radioactivity into TLFA of tissues 60 min later. The minimal flux of exogenous glucose carbon into FA was calculated as before (8).

**RESULTS**

**Rates of Body Glucose Carbon Replacement (Irreversible Disposal) and Conversion to FA and Other "End Products" in 24-hr-fasted and ad Libitum-fed Mice.** In order to calculate rates of FA synthesis from glucose carbon in animals that were not given glucose test meals, tracer glucose was injected i.v., and rates of FA synthesis in liver and carcass were calculated by semicompartamental analysis (4). First, we determined

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R. Kannan and N. Baker, unpublished observations.
whether the body glucose pool sizes were the same in the control and in the tumor-bearing mice. Although the mean plasma glucose concentration of fasted control mice differed significantly from that of the tumorous mice [control, 0.87 ± 0.10 mg/ml (n = 9); tumorous, 0.48 ± 0.09 mg/ml (n = 9)], the total body glucose values for the 2 groups, as measured by isotopic dilution at 60 sec (3), were very similar, i.e., 11.2 ± 1.1 mg in controls and 11.8 ± 1.9 mg in tumorous mice. The apparent discrepancy between the different plasma glucose concentrations and the similar total body glucose pool sizes is explicable in part by the hemodilution and the greatly enlarged extracellular volume found in these cancerous mice (10). Another factor that could have lowered the fasting plasma glucose concentration in the cancerous mice is malnourishment. Previous work has shown that mice bearing 8-day-old tumors consume 50 to 60% of the fat-free glucose diet as compared to controls (13).

The irreversible disposal rates of glucose calculated from the common glucose specific activity curve in Chart 1 was 260 

\[
\text{mg of glucose carbon per min per mouse for both control and tumorous mice (Table 1), which is consistent with values determined earlier for 24-hr-fasted mice (3). The percentage of injected [14C]glucose converted to TLFA in carcasses of control mice in the present study was about one-half of the value obtained before (8). This variability is probably due to differences in the actual fasting periods from one study to another.}
\]

As shown in Table 1, the flux of glucose carbon into TLFA in carcasses from tumorous mice was about one-half that of controls. In this dietary state, no significant difference between livers of control and tumor-bearing mice in the incorporation of glucose carbon into TLFA was observed. In each case, FA synthesis from glucose carbon in the livers of 24-hr-fasted mice was extremely low as compared to that in extrahepatic tissues (Table 1). Lipogenesis in the tumors of 24-hr-fasted mice was even lower than that in the livers of the hosts. About 3% of the FA synthesized from glucose carbon in the fasted tumor-bearing mice was made in the tumor.

Rates of FA synthesis from glucose carbon have been determined in mice 24-hr fasted, 24-hr fasted and refed a glucose test meal, and ad libitum fed and refed a glucose meal. A comparison of lipogenic rates (nmol of TLFA per min) in extrahepatic tissues from normal mice and mice bearing 8-day-old carcinomas is shown in Table 2. With some exceptions, rates of FA synthesis were generally lower in tumorous mice than they were in control mice. In the fasted-refed state, the rate of FA synthesis from glucose carbon in these tumorous mice was about one-third that of the normal mice; 30 to 40% of the FA newly synthesized from glucose carbon was made in the liver in both tumorous and control animals.

Total Rates of FA Synthesis in Livers and Extrahepatic Tissues of Fasted, Fasted-Refed, and Fed-Refed Control and Tumorous (8-Day Tumors) Mice. Rates of FA synthesis from [3H]O in carcasses of control and tumorous mice (Table 3) were not significantly different from each other in all 3 dietary states studied. However, in fed-refed mice, the total rate of FA synthesis in the whole body (carcass plus liver) was twice as great in control mice as it was in the tumor-bearing animals (658 nmol versus 340 nmol of TLFA per min per mouse). In both fasted control and tumorous mice that were refed a glucose meal, no significant lipogenic activation from 2-carbon units in the extrahepatic carcass was observed in contrast both to the marked activation observed from glucose carbon and to an earlier finding in control mice in which a doubling of the total lipogenic rate was found (8). The increase in lipogenic rates from 2-carbon units in the carcasses of the fed-refed control and the tumorous groups (4- to 5-fold over 24-hr-fasted mice) was not as great as was the increase (about 20- to 30-fold) seen with glucose carbon (cf. Table 2).

Several major differences between tumorous and control mice were found in the liver: (a) in the hepatic tissue of fasted tumorous mice, there was a significant increase in the rate of FA synthesis as compared to that in the controls; (b) this significant difference between livers of fasted control and tumorous mice disappeared when the fasted mice were refed a glucose meal. This reflected a 3-fold activation in livers of fasted-refed control mice and essentially no activation in livers of the tumorous mice. The differences in the fasting lipogenic activities and in the degree of activation in hepatic tissue of control and tumorous mice might suggest a subtle abnormality in the hepatic metabolic regulatory system in tumorous mice; (c) in the fasted-refed state, the rate of FA synthesis from [3H]O in livers of tumorous mice was significantly lower than was that of controls (315 nmol versus 293 nmol/min).

On the basis of the data in Table 3, we may also calculate the contribution of the liver to the total amount of FA synthesized in the whole body (carcass plus liver). Except for those in the fasted state, the livers of tumorous mice played the same relative role as they did in controls. Thus, in fasted-refed mice (control and tumorous), 25% of the total newly synthesized FA was made in the liver. In the fasted-refed state, this value increased to 40 and 45% in tumorous and control mice, respectively. The liver played a greater role in fasted tumorous mice than it did in fasted controls (28% versus 10%, respectively).

Lipogenesis from [14C]Glucose and [3H]O in Host Tissues of Mice Bearing 5-Day-Old Ehrlich Ascites Carcinomas. FA synthesis was examined in host tissues during an early stage of tumor development (Day 5) in fed-refed mice. The tumorous mice on Day 5 had the same food intake as did the controls. Although FA synthesis from glucose carbon appeared to be

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**Chart 1. Plasma glucose specific activity in 24-hr-fasted control mice and in mice bearing 8-day-old tumors.** [U-14C]Glucose was injected i.v. into unanesthetized control and tumorous mice, and plasma glucose specific activity was determined in blood samples collected at 1, 5, 15, 30, and 60 min after injection. A composite least-squares-fit curve is drawn through all data points, since specific activity data did not differ in control and tumorous mice. Bars, S.E.


Tumor and Host Tissue Lipogenesis in Ehrlich Ascites Carcinoma

Table 1

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control (μg glucose carbon/min/mouse)</th>
<th>Tumorous (μg glucose carbon/min/mouse)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incorporation into 14C-FA at 60 min (q2) (% injected [U-14C]glucose)</td>
<td>0.40 ± 0.052</td>
<td>0.19 ± 0.013</td>
</tr>
<tr>
<td>Incorporation into all end products at 60 min (q2) (% injected [U-14C]glucose)</td>
<td>0.0088 ± 0.0013</td>
<td>0.0108 ± 0.0012</td>
</tr>
<tr>
<td>Flux into TLFA (R2) (μg glucose carbon/min/mouse)</td>
<td>1.09 ± 0.14</td>
<td>0.52 ± 0.036</td>
</tr>
</tbody>
</table>

Table 2

<table>
<thead>
<tr>
<th>Dietary state</th>
<th>Carcass</th>
<th>Tumorous</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Tumorous</td>
</tr>
<tr>
<td>24-hr fasted</td>
<td>6.4 ± 1.2</td>
<td>3.1 ± 0.19</td>
</tr>
<tr>
<td>Fasted-refed</td>
<td>36 ± 6.4</td>
<td>34 ± 4.1</td>
</tr>
<tr>
<td>Fed-refed</td>
<td>195 ± 50</td>
<td>57 ± 13.9</td>
</tr>
</tbody>
</table>

Table 3

<table>
<thead>
<tr>
<th>Dietary state</th>
<th>Carcass</th>
<th>Tumorous</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Tumorous</td>
</tr>
<tr>
<td>24-hr fasted</td>
<td>82 ± 10</td>
<td>51 ± 7</td>
</tr>
<tr>
<td>Fasted-refed</td>
<td>80 ± 7.5</td>
<td>76 ± 7</td>
</tr>
<tr>
<td>Fed-refed</td>
<td>365 ± 66</td>
<td>205 ± 78</td>
</tr>
</tbody>
</table>

Table 4

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>Tumorous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incorporation into 14C-FA at 60 min (q2) (% injected [U-14C]glucose)</td>
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</tr>
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<td>0.0108 ± 0.0012</td>
</tr>
<tr>
<td>Flux into TLFA (R2) (μg glucose carbon/min/mouse)</td>
<td>1.09 ± 0.14</td>
<td>0.52 ± 0.036</td>
</tr>
</tbody>
</table>

DISCUSSION

A major objective in this study was to establish whether the commonly observed loss of body fat during cancer growth is due, in part, to a diminished capacity of the host’s tissues to

significantly depressed in the carcasses of tumor-bearing mice, even at this early stage of tumor growth, lipogenesis from glucose carbon in the livers of these mice was not inhibited (Table 4). Moreover, the total rates of FA synthesis from all 2-carbon units in both carcasses and livers of mice bearing 5-day-old tumors were not significantly different from those of controls. These results, when contrasted with the results in Tables 2 and 3 for fed-refed mice, suggest strongly that when tumorous mice have eaten normally they synthesize FA at about the same rate as do control mice.

Lipogenesis from Glucose Carbon and All 2-Carbon Units in Ehrlich Ascites Carcinoma. On the basis of the incorporation of [3H2O] and [U-14C]glucose into TLFA of the whole tumor (8 days) under varying nutritional states, we calculated rates of FA synthesis expressed as nmol of TLFA per min per 7 ml of whole tumor (Table 5). We found a 4-fold activation in lipogenesis from glucose carbon in the tumor when 24-hr-fasted mice were refed a glucose-rich meal. The lipogenic rate from glucose carbon in the tumor of fed-refed mice was 12-fold greater than was the rate in the fasted state. The total rate of lipogenesis in the tumor increased marginally in the fasted-refed state over the fasted state, but a significant 2-fold increase (p < 0.02) occurred in tumors of fed-refed mice. Lipogenesis in the tumor cell from glucose carbon formed an insignificant portion of total lipogenesis from 2-carbon units in the fasted or in the fasted-refed states. However, in 8-day tumors of fed-refed mice, glucose carbon contributed about 20% of the total carbon used for FA synthesis.

A major objective in this study was to establish whether the commonly observed loss of body fat during cancer growth is due, in part, to a diminished capacity of the host’s tissues to
synthesizes FA. Earlier work from this laboratory has shown that in mice maximal lipogenesis can be approached in daylight hours by using animals that are fed ad libitum and refed a small glucose test meal. This condition had not been used to study rates of FA synthesis in cancerous animals. In mice bearing 8-day-old tumors, FA synthesis from exogenous glucose carbon was 3 times faster in the control mice than it was in the tumor-bearing host in the fed-refed state (300 nmol versus 100 nmol of FA per min per mouse, liver plus carcass). The diminution in the total rate of lipogenesis from 2-carbon units in these cancerous mice was less pronounced (660 nmol versus 340 nmol of FA per min per mouse in control versus tumor-bearing mice, respectively), but the difference was still statistically significant (p < 0.005). Thus, defective lipogenesis within the host tissues of cancer-bearing animals probably plays an important role in the ultimate depletion of fat stores.

Diminished lipogenic rates in the host’s tissues in mice with 8-day-old tumors need not reflect a fundamental abnormality in the regulatory process. Indeed, the hepatic and extrahepatic tissues of tumor-bearing mice behave similarly to those of control mice with respect to activation and inactivation of lipogenesis by dietary manipulation. These changes were extremely large, especially with respect to glucose carbon and its relative role as a carbon source for FA synthesis; moreover, the behavior of the lipogenic enzyme systems in response to feeding and fasting was very similar to that reported recently in normal mice (8). Glucose feeding has been shown earlier to produce a prompt inhibition of FFA mobilization in mice bearing Ehrlich ascites carcinoma (1). Both the lipogenic and lipolytic regulatory mechanisms are known to be insulin dependent; therefore, this type of hormonal regulation seems to be functional in these mice. Despite the presence of the regulatory mechanisms, lipogenesis was still slower in mice bearing 8-day-old tumors than it was in control mice. This phenomenon is undoubtedly related to malnourishment which is known to occur in cancer-bearing mice fed ad libitum; decreased food intake is known to decrease the lipogenic rates in both liver and adipose tissue (8, 13). This was substantiated from experiments carried out with well-nourished mice bearing 5-day-old carcinomas. Rates of FA synthesis from all 2-carbon units in these mice were the same as they were in the controls.

The second objective of our study was the evaluation of lipogenic rates from both glucose carbon and all 2-carbon units in the cancer itself in vivo under several nutritional conditions. Mice bearing 8-day-old Ehrlich ascites carcinomas were used for most of these studies so that we would have sufficient tissue to work with and be able to compare the lipogenic rates with other parameters that we have established previously in mice bearing tumors at the same stage of growth. The rate of FA synthesis from glucose carbon was an order of magnitude faster in cancers growing in fed-refed mice than in those growing in fasted animals. However, glucose was a relatively minor carbon source for FA synthesis, especially in the fasted state where only 3% of the newly synthesized FA was derived from glucose carbon. The percentage rose to 20% in the fed-refed state, but the total rate of lipogenesis (based on 1H2O studies) was only twice as fast in the cancers of fed-refed mice as it was in those of the fasted animals. Thus, as in other tissues studied in both the host and in control animals, the regulation of lipogenesis in the cancer cell by dietary glucose involves a change in the sources of carbon for lipogenesis more than it does a change in the absolute rate of synthesis.

Both FFA and very-low-density lipoprotein triglyceride fatty acid have been proposed, on the basis of studies in vitro, as possible inhibitors of lipogenesis in Ehrlich ascites carcinoma (14-16). We do not know whether tumor extracellular fluid FFA concentrations undergo the marked fluctuations that occur in circulating FFA levels (1). Such rapid changes in the tumor fluid FFA concentration would not be expected, according to evidence on FFA transport obtained in our laboratory (10). However, we feel that we can rule out very-low-density lipoprotein triglyceride fatty acid as a major metabolic regulatory inhibitor of lipogenesis because we have shown in earlier studies that its concentration falls during fasting and rises on refeeding (11, 12). Thus, an inverse correlation between lipogenic rates and the lipoprotein levels in the tumor fluid does not occur.

By measuring rates of lipogenesis in the tumor in vivo under optimal nutritional conditions (fed-refed state), we should gain considerable insight regarding the relative roles of the host and the tumor itself in supplying the cancer cells with FA needed for growth and metabolism. However, such comparisons can only be made if one first establishes that the 3H and 14C found in the tumor FA under the conditions of our study were actually incorporated into FA by the cancer cells (rather than by the host cells) and then transported to the tumor cells. On the basis of our earlier published and unpublished studies, we think that most of the FA radioactivity found in the tumors in our present studies was synthesized by the cancer cells de
Tumor and Host Tissue Lipogenesis in Ehrlich Ascites Carcinoma

Tumor and Host Tissue Lipogenesis in Ehrlich Ascites Carcinoma

novo rather than by the host and then transported to the tumor. However, further studies are required to establish this point. The apparent rates of de novo FA synthesis in the cancers of fasted and fed mice ranged from 6 to 11 nmol of FA per min per 7 ml of tumor. This is almost identical to the value (10 nmol/min) reported for the rate of FFA transport from the circulation to the tumor (10, 18). Therefore, we conclude, contrary to the in vitro findings of earlier workers (17), that FA synthesis de novo may be as important a source of FA for the tumor as is the circulating FFA provided by the host.

ACKNOWLEDGMENTS

We wish to thank David Learn for his outstanding technical assistance.

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Dietary Control of Lipogenesis \textit{in Vivo} in Host Tissues and Tumors of Mice Bearing Ehrlich Ascites Carcinoma

Ramaswamy Kannan, Irving Lyon and Nome Baker


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