Differential Survival of Solid Tumor Cells after Inoculation into Established Ascites Tumors*

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It has been shown (6) that only a limited number of transplantable mouse neoplasms can be grown in the form of typical "ascites tumors," if the term is used to denote a condition where free tumor cells or cell complexes multiply in the peritoneal fluid, and, accompanied by the gradual accumulation of ascites, there attain a nearly pure culture. The 42 tumors surveyed showed three distinctly different patterns of behavior: (a) Six tumors, mainly malignant lymphomas, grew readily in the ascites form when adequate numbers of cells from the solid tumor were injected intraperitoneally. (b) Six other neoplasms yielded exudates poor in tumor cells. Upon further serial intraperitoneal transfer of these exudates typical ascites tumors were eventually obtained. (c) The majority, 30 tumors, did not lend themselves to transformation into the ascites form under the conditions used.

The present investigation represents the first step in a series of experiments designed to gain information about the factors concerned in determining whether a certain tumor can be grown in the ascites form. It is possible that the difference among these three tumor categories is dependent on a differential capacity of the various cell populations to survive and multiply in the peritoneal fluid. Alternatively, the findings could be due to variations in the capacity of different tumors to provoke primary exudate formation after intraperitoneal inoculation. In the latter case, survival of various tumor cell populations inoculated into established peritoneal exudates would not be expected to be systematically different. To decide among these possibilities, several neoplasms which have been studied previously with regard to their ascites tumor-producing capacity were inoculated as cell suspensions into established peritoneal exudates, and their survival in the fluid was tested by means of a biological assay.

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MATERIALS AND METHODS

Mice.—The following five inbred strains were used: C3H/St, A/St, C57BL, DBA, and C57L. Breeding pairs of the first four strains were obtained from Dr. T. S. Hauschka (Institute for Cancer Research, Philadelphia) in 1950. The C57L strain was kindly sent us by Miss E. Fekete of the Jackson Memorial Laboratory. All five strains were maintained by strict brother-sister mating. For part of the experiments, F1 hybrids produced from various combinations of the five strains were used. Animals of both sexes were employed, at an age of 2-5 months and with a body weight of 18-25 gm. The mice were kept on a standard pellet diet, which, as well as drinking water, was available ad libitum at all times.

Tumors.—Seventeen different transplantable mouse tumors were used (Table 1). Most of these neoplasms had been tested previously with regard to their capacity to grow in the ascites form (4, 6) by passing them through serial intraperitoneal transfers; peritoneal exudates were used for inoculation whenever possible.

The tumors capable of growing in the ascites form (see col. 7, Table 1) were maintained by serial intraperitoneal transfers of ascitic fluid. For the present experiments, 0.2 ml. was inoculated subcutaneously into mice of a compatible strain, and the developing solid tumors were used. The neoplasms that did not grow as ascites tumors were maintained by serial subcutaneous passages of cell suspensions prepared by mechanical disruption of the tissue.

In the present work medium-sized solid tumors of both kinds were removed, freed from grossly necrotic areas, minced, and disrupted by the centrifuge method of Hauschka and Poppe (5). Using a disc of stainless steel mesh, 60 wires/inch, and buffered physiological saline as medium, uniform suspensions were obtained which contained mainly single cells and small clumps of cells. The suspensions were diluted twentyfold by Tyrode's solution containing 0.05 per cent eosin, and the unstained cells were quickly counted in a Buerker hemocytometer. According to Schrek (9), this represents the number of viable cells. More recent evidence (1) indicates that, while unstained cells are viable, staining does not necessarily mean cell death. The number of unstained cells would therefore represent the minimum number of viable cells. A predetermined number of unstained cells (usually 1 million) was used for inoculation according to the experimental scheme as presented below.

The growth rate of the solid tumors was estimated on the basis of daily caliper measurements according to the method described by Schrek (8). Three different tumor diameters were measured, and the geometric mean was calculated and plotted against time. Approximately straight-line relationships were usually obtained on an arithmetic scale. Least square lines were fitted to the experimental points, and the regression coefficient was taken to indicate the average daily linear growth of the tumor.
RESULTS

The main technical problem was the production of sufficient amounts of peritoneal exudate which could be presumed to be to some degree similar in composition to the fluid in which ascites tumor cells usually grow. Although there are several ways to produce sterile exudates in the peritoneal cavity of mice, large amounts of fluid are seldom obtained, and it is difficult to maintain the exudate at a satisfactory level for an adequate period of time. Such exudates are usually produced by means of mechanical or chemical irritation, and they are not necessarily comparable to the medium of ascites tumor cells. It was therefore decided to utilize an established ascites tumor to produce and maintain a sufficient volume of exudate into which cell suspensions from the test tumor could be inoculated. Advantage was taken of the high strain specificity of two ascites lymphomas which were used for this purpose and which will be referred to as "carrier" tumors. The solid neoplasms tested will be designated as "test tumors."

The two carrier tumors, 6C3HED lymphoma and DBA lymphoma, were found to grow progressively in their strains of origin only (C3H and DBA, respectively), or in F1 hybrids between the original and any other strain (2, 5). In each experiment, test tumors were selected that had originated and could grow in a mouse strain different from that of the carrier tumor. Under such circumstances, the carrier and test tumors could be grown together in F1 hybrids between their two parent strains and separated by subsequent inoculation into animals of the two original pure lines.

Chart 1 illustrates the experimental procedure for carrier tumor 6C3HED and test tumor C1300. Five A × C3H F1 hybrids were given an inoculation in Step I of 0.2 ml. 6C3HED ascites on day 0. By day 8 the mice showed pronounced abdominal distension due to the accumulation of 3–4 ml. of ascites. Into this fluid a suspension containing 1 million eosin-unstained cells from a C1300 tumor was injected (Step II). On the 8th day the mice were killed, and all the ascitic fluid was collected.

<table>
<thead>
<tr>
<th>Designation</th>
<th>Type*</th>
<th>Year of origin</th>
<th>Derivation</th>
<th>Mouse strain of origin</th>
<th>Progressive growth in mouse strain</th>
<th>Transformation into ascites tumor†</th>
<th>No. of transfer generations before these experiments</th>
</tr>
</thead>
<tbody>
<tr>
<td>6C3HED</td>
<td>ind. lymphosarcoma</td>
<td>1941</td>
<td>thymus</td>
<td>C3H</td>
<td>C3H only</td>
<td>+i (1950)</td>
<td>&gt;100</td>
</tr>
<tr>
<td>DBA lymphoma (Dalton)</td>
<td>sp. lymphosarcoma</td>
<td>1947</td>
<td>thymus</td>
<td>DBA</td>
<td>DBA only</td>
<td>+i (1950)</td>
<td>&gt;100</td>
</tr>
<tr>
<td>Ehrlich</td>
<td>sp. carcinoma</td>
<td>1906</td>
<td>mammary gland</td>
<td>non-inbred</td>
<td>all strains</td>
<td>+i (1948)</td>
<td>&gt;100</td>
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<tr>
<td>150091a</td>
<td>sp. adenocarcinoma</td>
<td>1928</td>
<td>mammary gland</td>
<td>A</td>
<td>A; also some foreign strains</td>
<td>+g (1950)</td>
<td>&gt;100</td>
</tr>
<tr>
<td>Krebs 2</td>
<td>sp. carcinoma? reticulosarcoma?</td>
<td>1933</td>
<td>?</td>
<td>non-inbred</td>
<td>all strains</td>
<td>+g (1950)</td>
<td>&gt;100</td>
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<tr>
<td>TAs</td>
<td>sp. adenocarcinoma</td>
<td>1948</td>
<td>mammary gland</td>
<td>muscle</td>
<td>C3H</td>
<td>C3H</td>
<td>+g (1951)</td>
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<tr>
<td>MCI1</td>
<td>ind. rhabdomyosarcoma</td>
<td>1945</td>
<td>muscle</td>
<td>C3H</td>
<td>C3H: also some foreign strains</td>
<td>+g (1951)</td>
<td>&gt;100</td>
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<td>MC1M</td>
<td>ind. sarcoma</td>
<td>1946</td>
<td>muscle</td>
<td>C3H</td>
<td>C3H: also some foreign strains</td>
<td>+g (1951)</td>
<td>&gt;100</td>
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<td>Lymphoma EL. 4</td>
<td>ind. lymphoma</td>
<td>1945</td>
<td>infiltrated organs</td>
<td>C37BL</td>
<td>C37BL</td>
<td>+i (1952)</td>
<td>&gt;75</td>
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<td>SPAH</td>
<td>sp. adenocarcinoma</td>
<td>1950</td>
<td>mammary gland</td>
<td>A</td>
<td>A</td>
<td>-e</td>
<td>52</td>
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<td>SA9</td>
<td>sp. adenocarcinoma</td>
<td>1951</td>
<td>mammary gland</td>
<td>C3H</td>
<td>C3H</td>
<td>-e</td>
<td>54</td>
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<td>SSA</td>
<td>sp. adenocarcinoma</td>
<td>1952</td>
<td>mammary gland</td>
<td>A</td>
<td>A</td>
<td>-e</td>
<td>27</td>
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<tr>
<td>S6C</td>
<td>sp. adenocarcinoma</td>
<td>1952</td>
<td>mammary gland</td>
<td>C3H</td>
<td>C3H</td>
<td>-e</td>
<td>10</td>
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<tr>
<td>C954</td>
<td>carcinoma of liver parenchyma cells</td>
<td>1940</td>
<td>liver</td>
<td>C37L</td>
<td>C37L</td>
<td>-e</td>
<td>&gt;60</td>
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<td>SSD</td>
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<td>mammary gland</td>
<td>DBA</td>
<td>DBA</td>
<td>-0</td>
<td>11</td>
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<tr>
<td>S6C</td>
<td>sp. adenocarcinoma</td>
<td>1952</td>
<td>mammary gland</td>
<td>C3H</td>
<td>C3H</td>
<td>-0</td>
<td>6</td>
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<tr>
<td>C1300</td>
<td>sp. neuroblastoma?</td>
<td>1940</td>
<td>region of spinal cord</td>
<td>A</td>
<td>A; also some foreign strains</td>
<td>-0</td>
<td>&gt;100</td>
</tr>
</tbody>
</table>

* Sp. = spontaneous; ind. = induced.
† Successful immediately: +i; successful gradually: +g; unsuccessful, with regular exudate formation: -e; unsuccessful, with no regular exudate formation: -0; in parentheses: year of transformation.
and pooled. Five to ten mice of the two original mouse strains (C3H and A/ST, Step III) were then given intraperitoneal inoculations of 0.2 ml. of the fluid and were observed for solid tumor or ascites development. The tumors were examined histologically, and the available ascites was studied on stained smears. In this particular experiment it was found that all five C3H mice died with typical 6C3HED ascites after 8–12 days. All ten A/ST showed initial ascites formation followed by rapid regression, and they survived an observation period of 3 months without any evidence of tumor growth. This result was interpreted as indicating that very few or no living C1300 cells were present within the established 6C3HED ascites at the end of the 2 days' observation period in Step II. The majority of the cells can be assumed to have died or settled on the peritoneal surface.

A different result was obtained when testing the solid tumor that developed after the subcutaneous inoculation of TA3 ascites carcinoma. This neoplasm is also homologous to the A/ST strain, but, in contrast to C1300, is capable of growing in the ascites form and shows a characteristic proliferation of small tumor cell complexes in the peritoneal fluid. When performing the inoculations of Step III, it was found that ten C3H mice again died in 9–12 days with typical 6C3HED ascites. The ten A/ST mice showed initial accumulation of ascites (days 4–9), followed by decrease or disappearance of ascites (days 15–18), reappearance of ascites on the 18th–20th day, and death of the mice on the 20th–25th day. Cytological examination of the ascites (obtained by repeated punctures with a glass capillary) showed the following succession of events: development of 6C3HED lymphoma, regression of the lymphoma with vacuolization of the lymphoblasts, intense inflammatory reaction, and disappearance of the lymphoblasts. Simultaneously with the regression of 6C3HED, typical complexes of TA3 tumor cells began to appear and developed gradually into an almost pure culture (Figs. 1–4). This condition remained and was followed by ascites accumulation until the death of the animals. This experiment indicated that TA3 cells survived in the fluid medium of 6C3HED ascites in Step II.

Experiments were carried out in a similar way on all test tumors, with appropriate modifications of the mouse strains. Figures 5–8 show the cytological appearance of the peritoneal fluid at different times after inoculation of DBA lymphoma (carrier) containing surviving MC1M cells (test tumor) into C3H mice. Again, the initially apparently pure culture of lymphoma cells regressed, while MC1M cells gradually appeared and finally dominated the picture entirely.

The succession of events was essentially similar with all other tumors that survived Step II. The results are summarized in Table 2. A comparison of column 5 with columns 6 and 7 shows that tumors with ability to grow in the ascites form survived much better than those capable of solid growth only. Before concluding that various tumor-cell populations have a differential capacity for survival in the free cell form in the peritoneal fluid, it is necessary to exclude the possibility of a spurious correlation arising from differences in dose of inoculum, in growth rate, or in sensitivity to immunological reactions of the host. These sources of error will be considered briefly in the following.

**Chart 1.—Schematic representation of experimental design.** Carrier tumor: 6C3HED, homologous to the C3H mouse strain. Test tumor: C1300, homologous to the A/ST strain. 6C3HED was used to produce ascites in A × C3H F1 hybrids (Step I). After 6 days, cell suspension from C1300 solid tumor was inoculated into the ascites of the same hybrids (Step II). After 2 more days the hybrids were killed, and their ascitic fluid was inoculated into mice of the two original pure strains (Step III). For further details see text.

a) *Inoculum dose.*—The number of eosin unstained cells of the test tumor injected in Step II was 1 million in all experiments reported in Table 2. The percentage of tumor cells in the inoculated sample probably varies among different neoplasms. A possible higher proportion of tumor cells in the AS tumors could conceivably lead to their better survival after inoculation in Step II. Al-
though systematic differences between the two groups of neoplasms with regard to the proportion between stroma and tumor parenchyma were not apparent histologically or upon examination of stained smears of the cell suspensions, the following experiments were carried out to exclude this possibility: Three representative “negative” tumors were selected, namely, SPAH, S3A, and C1900, and their survival in 6C3HED ascites was tested by using 2 and 4 million unstained cells as inocula. Survival was not improved despite the increase in the size of the inoculum. It is therefore of the fact that they are intimately intermixed with the cells of the carrier lymphoma.

The group of “positive” tumors includes several neoplasms of low strain specificity, while the majority of “negative” tumors are highly strain specific (see Tables 1 and 2). It could be argued that the “positive” group might consist of tumors that are insensitive to the immunological reaction directed against cells of the foreign carrier tumor, while the “negative” ones are damaged by this reaction in a nonspecific way. Contrary to this assumption, it could be demonstrated that two of the

### Table 2

**Survival of the Test Tumors in the Ascites Produced by the Carrier Lymphomas**

<table>
<thead>
<tr>
<th>Test Tumor</th>
<th>Transplantation Line</th>
<th>No. Experiments</th>
<th>Daily Av. Linear Growth (mm.)</th>
<th>Growth in the Ascites</th>
<th>Survival After Inoculation</th>
<th>Comment</th>
</tr>
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<tbody>
<tr>
<td></td>
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</table>

The very improbable that the results could be due to systematic differences in tumor cell dose.

**b) Growth rate.—**Caliper measurements were carried out to study whether there is any major systematic difference between the two groups of test tumors with regard to growth rate. No such difference could be found (col. 4, Table 2).

c) **Differential sensitivity to the immunological reaction directed against the carrier tumor.**—In Step III, cells from the carrier lymphoma, intermixed with surviving cells of the test tumor, were inoculated into mice incompatible with the carrier but compatible with the test tumor. The carrier tumor began to grow in the foreign strain, but was soon attacked and finally destroyed by the immunological reaction of the host. To obtain a positive result with this experimental design, cells of the test tumor have to survive this reaction in spite of the fact that they are intimately intermixed with the cells of the carrier lymphoma.

The group of “positive” tumors includes several neoplasms of low strain specificity, while the majority of “negative” tumors are highly strain specific (see Tables 1 and 2). It could be argued that the “positive” group might consist of tumors that are insensitive to the immunological reaction directed against cells of the foreign carrier tumor, while the “negative” ones are damaged by this reaction in a nonspecific way. Contrary to this assumption, it could be demonstrated that two of the
applies for the combination of S6C and DBA mice.

S3D was tested in the main experiments by inoculation into C3HED as carrier (Step II), with DBA × C57 F1 hybrids. In Step III, the ascites was inoculated into pure DBA animals which destroyed the carrier cells of C57 origin. Conversely, tumor S6C of the C57 strain was tested by being mixed with the DBA lymphoma as carrier. In Step III, the surviving cells of S6C had to be left unharmed by the reaction of the C57 animals against the "carrier" cells of DBA origin. It was therefore questioned whether S3D cells could survive in DBA mice when, closely intermixed with tumor cells of C57 origin that were being gradually destroyed by the host; and, conversely, whether S6C cells could survive in C57 hosts that react against tumor cells of the DBA strain. To answer this question, the following experiment was performed:

Cell suspensions of S3D and S6C were prepared in the usual way and the number of viable cells counted. About 20 million cells from each tumor were mixed and inoculated subcutaneously to ten DBA/C57 F1 hybrids which support the progressive growth of both tumors. All ten animals developed large neoplasms 3-5 weeks after inoculation. Their tumors were further implanted into ten DBA and ten C57 mice. All animals developed tumors. The neoplasms that appeared in the DBA mice had the characteristic morphology of S3D and did not grow progressively in C57 mice. Conversely, the tumors of the C57 mice showed the morphological features of S6C and did not grow in pure line DBA mice. This proves that both tumors survived the procedure and were left unharmed by the immunological reaction directed against the admixed cells of the other, genetically incompatible neoplasm.

DISCUSSION

The present experiments have demonstrated that tumors unable to grow in the ascites form are also largely incapable of survival in the free cell form for 2 days within a pre-established peritoneal exudate. Only occasional survival could be demonstrated in a few cases (see Table 2). The refractoriness of these neoplasms to conversion into ascites tumors is thus probably determined by some nutritional or other characteristic of their cells which is incompatible with the environmental conditions prevailing in the peritoneal fluid. The alternate hypothesis, which would ascribe the behavior of these tumors to a defective capacity to elicit the primary accumulation of peritoneal exudate, is improbable.

The neoplasms that were adapted to growth in the ascites form showed, in general, a very good survival, as could be expected. The absence of tumors in a few animals in Step III can be explained as a probable dilution effect. Assuming the presence of 3-5 ml ascites in the mice used for Step II and survival of the test tumor cells at inoculum level, the order of magnitude of the number of neoplastic cells inoculated in Step III can be estimated as 4-6 × 10^4 at a maximum. Cell doses of this magnitude do not invariably lead to tumor development in 100 per cent of the mice.

It was surprising that no difference in survival could be found between the SS and AS lines, respectively, of the two sarcomas MC1A and MC1M. SS designates the original solid line, kept by serial subcutaneous transplants, while AS symbolizes solid tumors produced by subcutaneous inoculation of fluid from the established ascites form. Both sarcomas, although different with regard to morphological and growth characteristics, show similar behavior in the following respects:

The SS forms are not capable of growth in the ascites form immediately. Serial intraperitoneal passage of peritoneal exudate is necessary before a typical ascites tumor can be obtained (4). The number of such transfers was 24-26 for MC1A and ten to twelve for MC1M in our previous experiments. The AS forms, on the other hand, can give rise to typical ascites tumors in all animals after the first intraperitoneal inoculation of adequate cell numbers.

The production of ascites tumors is a matter of growth of free tumor cells in the peritoneal fluid, while the present experiments were designed to test cell survival only. As they failed to demonstrate any difference between the survival of AS and SS cells in the exudate, it must be assumed that the contrasting behavior of these lines depends on a differential capacity to grow in the fluid, while their cells can survive equally well, at least during the first 2 days after inoculation.

It would appear that there are at least three types of behavior with regard to the conversion of solid into ascites tumors. Certain tumor lines are apparently unable to survive within the peritoneal fluid, others can survive for some time but not grow, while still others can grow in the free cell form and reach a nearly pure tumor cell culture in the ascitic fluid.

There are some findings which suggest that tumor cells with the capacity to grow in the peritoneal fluid may arise by mutation (in the broad sense, not necessarily gene mutation). They can be briefly summarized as follows:

a) The neoplasms that could be converted into...
ascites tumors generally had an earlier date of origin than those that did not lend themselves to transformation (see Table 1). They are often more anaplastic, and their cells have passed through a much larger number of cell generations. Hence, the probability for the accumulation of mutations is greater.

b) With the exception of lymphomas, most of the successfully converted tumors had to be passed through serial exudate transfers (4) before they would grow in the ascites form. A small number of mutant cells possessing the capacity to grow in the fluid could be selected during such a procedure, as the cells surviving in the fluid are always the transmitters of the tumor.

c) Once established, the change is permanent without regard to the routine site of transplantation. The AS lines of the MCIA and Krebs 2 ascites tumors could be kept for at least eight and twenty subcutaneous solid transfer generations, respectively, without losing or diminishing the capacity to induce ascites tumors immediately.

The question remains what properties make tumor cells of certain types better suited to grow in the ascitic fluid than others. It can be speculated that at least part of the essential differences may be due to variations in the nutritional requirements. Warburg and Heipler (10) have shown that well developed ascites is very poor in free oxygen and glucose. Determinations showed a large decrease in the glucose content of the peritoneal fluid within 2 days after the inoculation of 2 million Ehrlich ascites tumor cells (E. Klein, unpublished). There is probably a similar shortage of many other important nutrients as well. Cells that are able to grow under such environmental circumstances may have less exacting nutritional requirements than the original cell population, and it is conceivable that they arise by an essentially nutritional type of mutation.

SUMMARY

When two transplanted tumors, specific for two different inbred strains of mice, are mixed and inoculated into F1 hybrid mice between the two original strains, both tumors grow progressively. By transplanting the resulting mixed neoplasm into mice of the original strains, the incompatible tumor regresses in each case, while the compatible tumor grows progressively and is unharmed by the reaction against its partner. This fact permitted the development of a technic for comparing the survival of various tumor cell suspensions within the fluid medium of two highly strain-specific ascites lymphomas.

Solid tumors that developed after the subcutaneous inoculation of eight different mouse ascites tumors were compared with eight other transplantable mouse neoplasms that were unable to grow in the ascites form. A difference was found between the two groups of tumors with regard to the survival of their cells within the pre-established lymphoma ascites for a period of 2 days. While the first group of tumors survived in most cases, cells from the second group very seldom survived. It is concluded that conversion of a solid neoplasm into an ascites tumor primarily depends on the capacity of its cells to survive and grow in the peritoneal fluid. The possible origin of such cells as nutritional mutants in the broadest sense is indicated.

ACKNOWLEDGMENTS

The authors wish to thank Mrs. Ulla Lomakka for valuable technical assistance.

REFERENCES

FIGS. 1-8.—Papanicolaou smears of ascitic fluid. Magnification X450.

FIGS. 1-4.—Ascites obtained by successive punctures from A/St mice inoculated in Step III (see Chart 1) with carrier tumor 6C3HED (specific for CSH strain) containing surviving cells of test tumor TA3 (homologous to A/St strain).

FIG. 1.—4 days after inoculation. Healthy 6C3HED lymphoma cells.

FIG. 2.—9 days after inoculation. Pyknotic and other degenerative changes in the lymphoma cells, appearance of macrophages and polymorphonuclear cells.

FIG. 3.—15 days after inoculation. No lymphoma cells left, polymorphonuclear cells dominate the picture. In the middle, a characteristic complex of two TA3 cells.

FIG. 4.—19 days after inoculation. The typical cell complexes of TA3 carcinoma are present in high concentration. The picture corresponds entirely to the usual appearance of the TA3 ascites tumor (see ref. 4, Figs. 19-21).

FIGS. 5-8.—Ascites obtained by successive punctures from C3H mice inoculated in Step III (Chart 1) with carrier tumor DBA lymphoma (specific for the DBA strain) containing surviving cells of test tumor MC1M (homologous to the CSH strain).

FIG. 5.—4 days after inoculation. DBA lymphoma.

FIG. 6.—10 days after inoculation. The picture is dominated by histiocytes with strongly vacuolized cytoplasm.

FIG. 7.—16 days after inoculation. Dilute culture of MC1M tumor cells.

FIG. 8.—20 days after inoculation. Nearly pure culture of MC1M cells, corresponding to the usual picture of the MC1M ascites tumor (see ref. 4, Figs. 36-38).
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