[CANCER RESEARCH 30, 1593–1597, June 1970]

Antigenicity of Cells Derived from Mouse Prostate Cells after Malignant Transformation *in Vitro* by Carcinogenic Hydrocarbons[1]

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SUMMARY

A line of cells derived from C3H mouse prostate was treated *in vitro* with methylcholanthrene and transformed to malignancy. Seventeen clones of transformed cells were tested for their immunogenicity in C3H mice. Mice were immunized by production of fibrosarcomas after injection of the individual clones; the tumors were ligated and regressed. Cells of the same clone were injected, and the numbers of tumors produced at different numbers of cells were compared with those induced in nontreated control mice. Eleven of these clones were definitely antigenic and only one was nonantigenic. No cross-reactivity was found within seven pairs of clones obtained from the same dish or within three clones derived from three different dishes. Thus, these clones derived by *in vitro* transformation with methylcholanthrene had multiple and distinct transplantation antigens. Immunization of mice with the nonmalignant control cells did not prevent the production of tumors on inoculation of transformed cells.

INTRODUCTION

It is now well established that sarcomas and carcinomas induced in mice and rats by carcinogenic hydrocarbons exhibit transplantation antigens (7, 9, 16, 20–22, 26, 29, 33–35). Similar antigens have been detected in hydrocarbon-induced papillomas (18) and azo dye-induced rat hepatomas (1). By and large, the hydrocarbon-induced sarcomas exhibit a low degree of cross-reactivity (16, 22, 28), but the total number of individual antigens is not now known. This field has been critically reviewed by Klein (15) and Prehn (23–25). In contrast to the large number of individual antigens of the chemically induced tumors, many of the tumors induced by many oncogenic viruses have common transplantation antigens (8, 13, 15, 31, 32).

For some time we have been engaged in the development of an *in vitro* system for carcinogenesis with hydrocarbons (3, 4, 11, 12), which has been made quantitative (5). Cell lines derived from adult C3H mouse ventral prostate grow in monolayer culture, reach a saturation density, stop growing, and do not cause tumors on inoculation into irradiated C3H mice (3). On treatment with carcinogenic hydrocarbons, these cells do not reach a saturation density, but pile up and cause sarcomas on injection into nonirradiated C3H mice (4). These sarcomas are lethal and transplantable and they metastasize. There is a quantitative relationship between the carcinogenic activity of hydrocarbons and the frequency of transformed colonies (5), and experiments have been presented to show that *in vitro* carcinogenesis can be carried out on single cells (19).

It is of considerable interest to determine whether the cells transformed *in vitro* with carcinogenic hydrocarbons are antigenic and, if so, whether they are cross-reactive. This paper reports initial studies along these lines. Studies on the antigenicity[5] of cells transformed *in vitro* with chemicals have not been published by other workers in the field (2, 6, 17, 30).

MATERIALS AND METHODS

Cell Cultures. Cell lines 25, 37, 42, and 80 were derived from organ cultures of C3H mouse prostate that had been treated with carcinogenic hydrocarbons, were permanent and malignant, and were cultured as described (11, 12). All had been maintained in culture for at least 1 year before these experiments were initiated. C-2 cells were obtained by methylcholanthrene transformation in mass culture (4) of a line of cells derived from C3H mouse prostate (3). A number of dishes containing B1 cells (defined in Ref. 3) were treated with various concentrations of methylcholanthrene maintained in the medium for 6 days: lines 6A, 6C, 4A, and 4C, 0.5 μg/ml; lines 3A, 3B, 2B, and 1A, 1.0 μg/ml; lines 8B, 7A, 7C, 5.0 μg/ml; and lines 10A, 10B, 9A, and 9B, 10.0 μg/ml. After 2 weeks, individual piled-up transformed colonies (2 from each dish) were ring-isolated (27) and cultured individually. By this means, 17 different clonal lines

[1]This work was supported in part by Grant CA 7175 from the National Cancer Institute, NIH.

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Received August 25, 1969; accepted January 26, 1970.

[5]Throughout this paper the words "antigenicity" and "immunogenicity" are used as synonyms.

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of transformed cells, all derived from the same control cells, were obtained. The letters refer to clones derived from individual piled-up transformed colonies within a dish of that number (*i.e.*, 9A and 9B are 2 transformed clones derived from Dish 9). An equal number of dishes of the B1 cells, taken at the 25th subculture (3), was treated with 0.5% dimethyl sulfoxide, the solvent used for the methylcholanthrene, and none gave rise to any piled-up colonies.

Immunization Methods. All animals used were adult male C3H mice, obtained from A. R. Schmidt and Co., Madison, Wis.

In the 1st series of experiments (Table 1 and 2), 10^6 cells were injected i.m. into the right leg of C3H male mice. After the tumors had grown to about 20 mm in diameter (usually about 2 to 3 weeks), the legs were amputated as described by Karrer *et al.* (14). One week later, these mice were challenged by the injection of different numbers of cells from various lines. The control mice with no tumors also had a leg amputated.

Table 1

Antigenicity of long-term cell lines derived from C3H mouse prostate by treatment in vitro with carcinogenic hydrocarbons
The same cell lines were used for immunization and challenge.

	No. of cells injected					
	10^6		10^5		10^4	
Cell line	I[a]	C[b]	I	C	I	C
25	4/4[c]	5/5	2/5	5/5	0/5	2/5
37	6/9	7/9	2/5	2/4	2/5	2/4
42	7/7	5/6	1/5	4/5	0/5	2/5
80	5/5	4/4	5/5	5/5	5/5	5/5
C-2	6/6	6/6	3/5	5/5	1/5	0/4

[a] I, immunized mice.

[b] C, control, nonimmunized mice.

[c] First number is the number of tumors; the 2nd is the number of mice injected.

Table 2

Cross-reactivity investigation of the immunogenic lines of cells studied in Table 1
This experiment was carried out at the same time as those in Table 1. Therefore, Table 1 serves as the control for Table 2.

		No. of cells injected					
		10^6		10^5		10^4	
Immunizing cell line	Challenging cell line	I	C	I	C	I	C
25	42	4/5	5/5	5/5	6/6	2/8	6/9
42	25	4/5	4/5	4/6	6/6	0/5	1/5
25	C-2	6/6	6/6	4/4	5/5	1/5	3/5
C-2	25	2/5	5/5	5/10	4/10	1/4	0/4
42	C-2	6/6	6/6	4/5	5/5	2/5	5/5
C-2	42	5/5	5/5	4/5	3/5	2/5	4/5

In the remainder of the experiments (Tables 3 to 5). C3H male mice were injected s.c. in the right flank with 10^6 cells of the different clonal lines transformed *in vitro*. When the tumors had reached about 15 mm in diameter, the bases were ligated with silk thread. During the next 2 weeks the tumors necrosed and dried up, leaving a scar at the site. One week later, these mice were challenged by inoculation s.c. in the left flank of different numbers of the clonal cell clines. At the same time, an equal number of control mice were also injected with the corresponding number of the same cells. All mice were observed for the presence of tumors for more than 100 days.

RESULTS

Immunogenicity of Cell Lines Derived from Carcinogen-treated Organ Cultures. In the experiments shown in Table 1, 5 long-term lines of transformed cells were tested for their transplantation antigenicity at 3 different logs of cell number. In each case, cells from the same line used to immunize the mice were injected into control and immunized mice and the tumor takes were noted. It is evident (Table 1) that cell lines 25 and 42 are antigenic, because there were significantly fewer takes in the immunized mice than in the nonimmunized ones at the proper cell inoculum. When too many cells were injected in the challenge, no difference in takes between the 2 groups was observed. From Table 1 it can be also seen that lines 37 and 80 were not antigenic under the conditions of our experiments, and line C-2 was of questionable antigenicity. There was also some variation of malignancy among these lines.

Table 3

Immunogenicity of individual clones transformed in vitro by methylcholanthrene
The same clones were used for immunization and challenge.

	No. of cells injected							
	10^5		5×10^4		10^4		5×10^3	
Clone No.	I	C	I	C	I	C	I	C
10A	2/2	8/8	1/5	4/8	0/4	0/4		
10B			1/3	3/4				
9A	0/1	6/6	0/3	2/4	0/4	1/3		
9B			0/4	3/4				
8B			1/5	5/5	0/5	0/5		
7A	1/3	4/5	0/3	4/4				
7C			4/7	7/7	0/4	0/4		
6A			0/2	2/4				
6C	3/3	6/6	0/3	4/5	0/3	2/3		
5B	3/6	3/6	1/7	2/7				
5C	3/3	6/6	4/4	4/4	3/6	5/6	0/3	2/3
4A			3/3	8/8	0/4	3/5		
4C					4/6	6/6	3/4	4/4
3A			0/5	4/5				
3B			3/4	3/4	0/3	2/3		
2B	1/5	3/5						
1A	1/3	3/5	0/4	2/5				

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Table 4

Test of possible cross-reactivity of immunogenic cell clones studied in Table 3

This experiment was done simultaneously with that of Table 3. Hence, Table 3 serves as the control for Table 4.

Immunizing clone	Challenging clone	No. of cells injected: 10^5 I	10^5 C	5×10^4 I	5×10^4 C	10^4 I	10^4 C
10A	10B			5/5	5/5	1/4	1/4
10B	10A			6/9	6/9		
9A	9B			4/4	5/6		
9B	9A			3/3	5/6		
7A	7C			2/4	2/4		
7C	7A			4/5	5/5		
6C	6A			2/3	2/4		
6A	6C			2/5	3/5		
5C	5B			1/4	1/4		
5B	5C					2/5	2/5
4A	4C					2/3	6/6
4C	4A					2/7	3/7
3A	3B					4/5	4/5
3B	3A			4/5	4/5		
2B	1A			3/4	3/4		
1A	2B	1/4	1/4				
1A	8B	4/4	4/4				
8B	1A			4/4	3/4		

The question of cross-reactivity was investigated in the experiments summarized in Table 2. Here, the 3 antigenic tumors were studied for cross-reactivity in all combinations, again at 3 different logs of cell numbers. It was found that lines 42 and C-2 were possibly cross-reactive in experiments where line 42 cells were used to immunize and line C-2 cells were used to challenge, and vice versa. A more ambiguous result was obtained with cells 25 and 42: when immunization was done with line 25 cells and challenge was with line 42 cells there was a small degree of cross-reactivity; however, when the line 42 cells were immunizing and were challenged with line 25 cells, there was no cross-reactivity. The same ambiguous result was found with cells 25 and C-2, although the line C-2 cells were very weakly antigenic.

Immunogenicity of Individual Clones Transformed *in Vitro* by Methylcholanthrene. A series of 17 transformed clones, including 7 pairs of clones derived from the same dish, were tested for immunogenicity as soon as enough cells were obtained. In these and subsequent experiments, the ligation instead of the amputation technique was used for the removal of the immunizing tumors. The results are shown in Table 3. In each case the same clone of cells was used to immunize and challenge. These data show that in order to demonstrate immunogenicity it is necessary to utilize the correct number of cells in the challenge. Therefore, it was often necessary to use several numbers of cells for the inocula. It was found under these conditions that in our judgment the following clones were antigenic: 10A, 9A, 9B, 8B, 7A, 6C, 5C, 4A, 3A, 2B, 1A. Clone 5B appeared to be nonantigenic, and the data were questionable on the antigenicity of clones 10B, 7C, 6A, 4C, and 3B. Thus, only 1 out of 17 clones clearly was not antigenic in this series of experiments.

An experiment was then set up to determine whether, in the same experiment as shown in Table 3, the pairs of individual clones derived from a single dish were cross-reactive. As shown in Table 4, each clone within the pair from the same dish was used both to immunize and challenge the mice. The results were quite definite. Of the 7 pairs of clones from the same dish, none was cross-reactive, 6 were definitely not cross-reactive, and with 1 (4A and 4C) the data were equivocal. In 3 pairs of individual clones from different dishes, there was no cross-reactivity. An experiment to test the possible cross-reactivity of all possible combinations of the clones derived from different dishes would be prohibitively large.

In an effort to determine whether the immunogenicity of the cells could be demonstrated before malignant transformation was carried out *in vitro* the following experiment was performed. The B1 control cells (from which the 17 clones were derived by *in vitro* treatment with methylcholanthrene) were injected at 10^5 cells i.p. at weekly intervals for 3 weeks, and 1 week later 5×10^4 8B cells were inoculated into the treated and control mice. The tumor takes were 3/4 and 4/4, respectively, showing that the control cells had no immunogenicity, at least to the cell clone used for the challenge and at the cell numbers used.

DISCUSSION

We have clearly demonstrated that individual clones of mouse prostate cells transformed to malignancy *in vitro* with methylcholanthrene are antigenic, in that (at the proper cell number) they do not produce tumors in mice in which a tumor produced by the same clones had been ligated and regressed. This finding parallels the situation that has been amply documented in *in vivo* hydrocarbon-induced sarcomas (7, 9, 15, 16, 20–23, 25, 26, 29, 33–35). The fact that 2 malignant clones within the same dish were noncross-reactive is quite analogous to the situation *in vivo*, where 2 tumors in the same mouse induced by the same hydrocarbon had distinctly different antigens (9).

The aforementioned experiments were carried out in such a way that the immunization of the mice was done within 2 weeks after the clones were isolated; the 2 weeks was required to grow enough cells to be injected into the mice and produce tumors. The very low antigenicity observed in the initial experiments (Tables 1 and 2) may be a consequence of the fact that they were carried out on malignant cells that had been maintained in continuous culture for at least 1 year. Also, the ligation technique probably produces better immunization than does amputation.

In the experiments on the cloned cells, no cross-reactivity was detected in 6 pairs of clones derived from the same dishes. Nor was cross-reactivity found when 3 pairs of clones from different dishes were studied. The cross-reactivity that was observed (Table 2) in the initial experiments with long-term lines may be discounted, in our opinion, on the

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basis that they were long-term lines in which the antigenicity had diminished and that they were not clonally derived.

Thus, our *in vitro* experiments are in good accord with the vast *in vivo* experience that, by and large, chemically induced sarcomas in mice are not cross-reactive, although some cross-reactivity has been reported (28). This fact lends support to the validity of our system as a model of carcinogenesis. The total number of different antigens in chemically induced sarcomas is not known, nor do we know the total number of different transplantation antigens in our *in vitro* hydrocarbon-induced malignant cells.

The acquisition of the antigen occurred concomitantly with the malignant transformation *in vitro*, because control nonmalignant cells failed to immunize mice against tumors induced by cells transformed from them *in vitro*.

At the time this work was started, it was widely believed that, in contrast to the situation of multiple distinct antigens in chemically induced tumors, most of the tumors induced by oncogenic viruses shared common transplantation antigens (8, 13, 15, 31, 32). Hence, we thought that if we were to find a lack of cross-reactivity in our transformed cells, it might provide evidence for the lack of participation of an oncogenic virus in our chemical transformation system. However, this approach was nullified by the subsequent experiments of Vaage (36) and Vaage *et al.* (37), who found nonvirus-specific transplantation antigens in spontaneous virus-induced mouse mammary carcinomas. Some of these antigens were noncross-reactive.

Some negative evidence on the participation of oncogenic viruses in our system has been provided by the following facts: (*a*) cell-free extracts of transformed cells were not cytopathic to various other cell lines (11); (*b*) cell-free extracts of transformed cells have not induced any tumors or neoplastic disease after inoculation into newborn C3H mice observed throughout their lifetimes (12); (*c*) few virus-like particles have been seen in electron micrographs of control and transformed cells (S. Mondal, P. T. Iype, L. M. Griesbach, and C. Heidelberger, unpublished data); and (*d*) Dr. J. W. Hartley and Dr. R. J. Huebner have failed to detect the group-specific antigens of the murine leukemia-sarcoma complex of viruses in our control and transformed cells either by direct complement-fixation tests or with their COMUL (10) test (R. J. Huebner, private communication). Although negative experiments cannot constitute proof, there is no evidence at present to indicate that the activation or "switching on" of a latent oncogenic virus is involved in our system for chemical carcinogenesis *in vitro*. Further studies along these lines are contemplated.

The origin of the multiple and distinct transplantion antigens in chemically induced tumors, both *in vivo* and now *in vitro*, represents one of the most intriguing problems in chemical carcinogenesis and at present defies theoretical explanation.

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