IMMUNOLOGY

Immunization of the *A/Jax* Mouse with Irradiated Cells of its Indigenous Tumour, Sarcoma I

ONE of the transplantable tumours widely used in investigations of tumour immunology is Sarcoma I (SaI), a tumour that originated in 1947 in a mouse of the Strong *A* strain that had been treated with dibenzanthracene[1]. We, as well as others, have maintained the tumour by transplantation in mice of the *A/Jax* strain, a sub-line of the Strong *A* strain, and utilize animals of this strain as the susceptible host in various experiments. Since any immunological response, even though it may be insufficient to cause rejection of the tumour, may influence the results of experiments with this tumour–host system, it appeared desirable to determine whether an immunological response to SaI occurs in the *A/Jax* mouse. From work of others it appeared that the possibility of an antigenic disparity between the tumour and the *A/Jax* mouse is favoured by the likelihood of genetic change in the mouse or the tumour during the prolonged period of transplantation and by the fact that the tumour was induced by dibenzanthracene[2,3].

Experiments were designed to demonstrate immunity to SaI in adult *A/Jax* animals by treatment with irradiated SaI ascites tumour (SaI-AT) prior to challenge with a lethal dose of SaI cells. (The *A/Jax* mice used were obtained in 1958 from the Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine. They were maintained in our laboratory by colony inbreeding for seven generations before use.)

The irradiation of SaI-AT cells was accomplished by X-ray treatment of adult *A/Jax* mice bearing an ascites tumour transplanted seven days earlier. Irradiation was delivered by a cobalt-60 source at a rate of 200 r./min. and a distance of 35 cm. The field size was 10 cm. × 10 cm. and the total exposure doses ranged from 18,000 r. to 36,000 r. (Dr. R. G. Parker assisted in the radiology).

Table 1. IMMUNITY TO SARCOMA I INDUCED IN *A/Jax* MICE WITH IRRADIATED TUMOUR CELLS

Route of administration of irradiated cells	Days after challenge with live SaI-AT cells 10	20	30	40	50	100	MST*
Intraperitoneal	0/12†	1/12	6/12	7/12	8/12	8/12	27·6
Intradermal	0/12	2/12	6/12	9/12	10/12	10/12	28·7
Untreated controls	0/14	14/14					17·1

* Mean survival time
† The numerator designates the total number of animals dead of tumour at the interval shown and the denominator the total number of animals tested.

Immediately after irradiation, the ascites fluid was removed with a 26-gauge needle and the tumour cells were counted. Ten million irradiated cells were injected into each of 12 female and 12 male adult *A/Jax* mice. The cells were deposited either intraperitoneally or intradermally in multiple sites of the skin and foot pads.

Fifty days after the administration of the immunizing dose of irradiated tumour cells all animals were given an intraperitoneal challenge dose of 1,000 viable ascites tumour cells. This number of tumour cells was equal to at least 100 lethal doses.

The data presented in Table 1 show that whereas all the control mice died of tumour before the 21st day many of the immunized mice survived for longer periods. Six of the 24 animals in the latter group were alive and healthy on the 100th day following challenge and at death showed no indication of tumour. The mortality-rates and survival-times of the immunized mice were not significantly different in female and male animals or in animals immunized by the two routes.

The results show that irradiated SaI-AT cells are immunogenic for the *A/Jax* mouse and indicate that antigenic differences exist between the *A/Jax* mouse and its indigenous tumour Sarcoma I.

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JOHN W. JUTILA*
RUSSELL S. WEISER
CHARLES A. EVANS

Department of Microbiology,
School of Medicine,
University of Washington, Seattle.

* Postdoctoral research trainee (*CRT*-5040).

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Staphylococcal Infection and Blood Groups

MANY workers have shown similarities between blood group substances and a variety of bacterial and non-bacterial antigens. Illchmann-Christ and Nagel[1] found that anti-A antibody was removed from B and O sera by *Staphylococci* and reasoned that *Staphylococci* possessed an A-like receptor. This suggests that persons of blood group A may have an increased susceptibility to staphylococcal infection due to an inability to elaborate an antibody which may cross-react with their own red blood cells.

Anderson, Coulter and Keynes[2] reported an extensive epidemiological investigation into staphylococcal infection and nasal carriage among mothers, babies and staff in a maternity hospital. No attempt was made at the time to correlate blood group with other inquiries, but this has now been done retrospectively.

All blood groups of mothers were recorded. Each mother was nose swabbed five times between the 36th ante-natal week and the 6th post-natal week. At the visit on the 36th week, each mother was questioned about previous staphylococcal infection and at the post-natal visit inquiries were made concerning infection following discharge from the hospital. The results are presented in Table 1.

Table 1

	Blood group of mother A	B	AB	O
Nasal carrier	127	22	7	135
Not nasal carrier	207	62	19	193
Positive history of staphylococcal infection	77	22	7	98
Negative history of staphylococcal infection	178	42	13	149
Staphylococcal infection in baby	29	15	0	37
No staphylococcal infection in baby	170	36	13	158

This table may be summarized: nasal carriage, O > A* > AB > B; history of staphylococcal infection, O > AB > B > A* < O.
* Indicates significance (χ^2 test $P < 0{\cdot}05$).

Group O persons are seen to be at a slight disadvantage both as regards nasal carriage and overt infection. It is doubtful whether these slight differences are of any clinical significance.

J. R. COULTER

Institute of Medical and Veterinary Science,
Adelaide.

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