selectively agglutinates reticulocytes and this agglutinating activity is inhibited by transferrin[5]. There does not seem to be any relationship between the reaction reported here and the reticulocyte HL-A antigens which are also present on leucocytes though absent on erythrocytes[6].

Our results indicate that guinea-pigs immunized with partially purified rabbit reticulocytes produced antibodies that were reactive with both rabbit erythrocytes and reticulocytes. When these antisera were absorbed with erythrocytes until no agglutinating or PCA activity could be detected, considerable activity against reticulocytes remained. Preliminary experiments using these antisera and others obtained against mouse reticulocytes show that the antisera also react specifically with the more immature erythroid cells of bone marrow.

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Rejection of Skin Grafts from Mice chronically infected with Lymphocytic Choriomeningitis Virus by non-infected Syngeneic Recipients

THE interaction of lymphocytic choriomeningitis (LCM) virus with mouse tissues has been discussed recently[1–5]. The fulminating disease, which occurs in adult mice after intracerebral inoculation with LCM virus, can be ameliorated by several treatments known to impair the immune response of the host, such as X-rays[6], amethopterin[7], thymectomy[8] and anti-lymphocyte serum[9]. The pathology of the disease[10] supports the current view that an immune reaction by the host against the virus or a virus product, located in or on infected cells, plays an essential part in its pathogenesis. A similar mechanism may be important in the chronic autoimmune-like disease in virus carrier mice[2,5]. Further elucidation of this disease mechanism depends on understanding the nature of the immune response and its target. A question of considerable interest is what the consequences might be for virus infected cells in the event of an immune response against the virus or a virus product. Here we report preliminary experiments involving transplantation of cells from virus carrier mice to syngeneic non-infected recipients which suggest that tissues of mice chronically infected with LCM virus undergo an antigenic change which can be detected by transplant rejection.

We used inbred mice of the strain SWR/Jax, 8–10 weeks old. Chronic LCM virus carrier animals were established by intraperitoneal inoculation of young mice less than 24 h old with 10^3 LD_{50} doses of the CA 1371 strain of the virus. Skin grafting was carried out essentially as described by Brown[11]. The skin of the experimental animals was lifted up in a mid-dorsal longitudinal fold and two circular grafts were removed by means of a steel punch, 9 mm in diameter. If the mice were to serve as graft recipients, the resulting two dorsolateral wounds served as graft beds without further preparation. Each recipient received an autograft and an isograft.

Table 1. RESULTS OF GRAFTING OF SKIN FROM LCM VIRUS INFECTED OR NON-INFECTED DONORS TO NON-INFECTED RECIPIENTS*

	Isografts	Autografts
Noninfected isograft donors	14/14	14/14
Infected isograft donors	0/13	4/13

* Denominator: number of grafts; numerator: number of successful grafts.

The mice each received two grafts: an autograft and an isograft from either an infected or a non-infected donor. All the recipient mice were non-infected. The pattern of successful and rejected grafts was clearly established on the fourteenth day after transplantation and did not change within the following 18 weeks of observation.

The results of experiments involving transplantation of skin from infected or non-infected donors to non-infected recipients are summarized in Table 1. They show that transplantation of skin from infected donors resulted consistently in rejection of the grafts. The rejection was clearly evident on the tenth day after grafting, and progressed rapidly to necrosis of the graft by the fourteenth day. In a number of cases, the autografts of animals receiving infected isografts were also rejected. Isografts between non-infected animals were uniformly successful. No sign of rejection was noted during 18 weeks after grafting.

The exact nature of the mechanism of graft rejection is unknown, but it is likely to be an immune phenomenon. Support for this view has been provided by the results of rechallenge with infected grafts of recipients that previously had rejected infected grafts. When inspected on the sixth day after grafting, these animals showed evidence of accelerated rejection of the infected skin characteristic of second set graft rejection. Control recipients that previously had received non-infected grafts did not differ from recipients that previously had not been grafted in their pattern of rejection of infected grafts. A likely explanation of the observed rejection of autografts by animals receiving infected isografts might be cross-infection of the autograft from the infected isograft, the two grafts being located no more than 10–15 mm apart.

Our results suggest that LCM virus produces an antigenic change in tissues of chronically as well as acutely infected mice detectable by tissue transplantation. Recent electron microscopic investigations of LCM virus infected cells[12] have shown that the virus lodges in microvilli of the cytoplasmic membrane. Such a location of the virus could account for the apparent histoincompatibility between skin of infected donors and non-infected syngeneic recipients. Histoincompatibility of a possibly similar nature has been reported by Breyere and Williams[13] between mice infected with a leukaemia virus and their syngeneic non-infected counterparts.

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