Thus, our preliminary observations show that: (1) early mortality and neurological manifestations of LCM virus infection are reduced in mice undergoing GVH reaction; and (2) the characteristic runting syndrome of GVH mice is precipitated and aggravated by LCM infection.

The data presented substantiate our earlier observations on decreased immunological reactivity of GVH mice[5,6]. Further, it seems that development of active neurological symptoms and early mortality in LCM-infected mice are closely correlated to availability of active lymphocytes. In our series, no such manifestations were noted on the height of GVH reaction when the lymphocyte count was the lowest. This is in accordance with the recently published observation of others that early mortality of LCM infection can be prevented by treatment with anti-lymphocyte serum[20]. The fact that in our present study the LCM infection enhanced and aggravated the runting syndrome established by the GVH reaction, is also in compliance with our earlier experiments made on neonatally thymectomized mice. Here, too, the runting syndrome elicited by thymectomy was considerably aggravated by LCM infection[16].

Our observations favour the hypothesis that runting or wasting is a non-specific syndrome, which may be caused by a variety of factors, among them viruses, in organisms with impaired immunological competence.

Mortality and incidence of neurological symptoms in mice undergoing graft-versus-host reaction and infected with virus of lymphocytic choriomeningitis

Days after LCM infection	5	6	7	8	9	10	11	12	16	17	24	37
No. of mice succumbed	4	1	4	4	3	2	2	3	2	1	1	1
No. of mice showing neurological symptoms	2	0	0	3	3	1	1	0	0	0	0	0

Résumé. Chez des souris hybrides F_1 adultes, subissant la maladie homologue produite par l'injection de cellules parentales, la mortalité précoce et les manifestations neurologiques d'une infection due au virus de la choriomeningite lymphocytique étaient réduits. D'autre part, le développement des symptomes caractéristiques du «runting» apparaît comme accéleré et aggravé par l'infection produit par ce virus.

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A Study on the Immunological Properties of Human Uterine and Placental Contractile Protein by Immuneadherence[1]

In 1965, King and Gröschel-Stewart isolated and characterized a contractile protein from human term placenta (PCP)[2] that exhibited properties similar to both human skeletal actomyosin (SAM) and the uterine contractile protein (UCP) described by Needham[3]. Several reports on the immunological behaviour of contractile proteins from various sources were published since[4–6]; and these studies suggested antigenic differences between skeletal, cardiac and smooth muscle contractile protein. Antiserum prepared by us against human UCP did not produce a reaction with SAM; however, precipitation did occur with both UCP and PCP under the indication of identity bands (Figure). In agreement with the previous investigators, we obtained multiple band formation. On the other hand, antiserum against highly purified SAM did not react with UCP; although a single precipitation band was obtained with the homologous antigen[7]. Attempts to establish a quantitative relationship between the above mentioned antigens and the antiserum by precipitation reaction[8] were inconsistent and therefore unsatisfactory; apparently not only due to the heterogeneity of the antigens used, but also due to the high ionic strength required for antigen solubility, which reduces precipitation rate of antigen-antibody complexes[9]. We therefore felt that the principle of immuneadherence (IA), extensively studied by Nelson[10,11], would offer a method suitable for demonstrating large molecular weight antigens, and a possibility of semiquantitative determination of the antigenicity of the proteins studied. IA has been described as a specific immunological reaction wherein particulate and soluble antigens sensitized with antibody and complement become attached to the surface of human erythrocytes, due to the presence of an IA receptor on the membrane of the erythrocyte. Utilizing this method, we were able to establish the immunological relationship between UCP and PCP.

The antigens (AG), actomyosin from surgical specimens of human striated muscle SAM, UCP and PCP were isolated as described elsewhere[2,12]. Antiserum (AS) was obtained from rabbits immunized according to Fink[4]. The animals were bled by cardiac puncture. The antiserum was absorbed 4 times with the same type human

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Titration of UCP + PCP (100 μg/ml) with rabbit antiserum against UCP after absorption of the antiserum with human erythrocytes

UCP Antiserum dilutions	1/200	1/400	1/800	1/1600	1/3200	1/6400	Controls
AS absorbed 4 ×							
AG + AS + C′	4+	4+	3+	2+	1+	1+	AG + C′ + E: ± AG + E: 0
AS + C′	2+	1+	±+	±+	±	±	C′ + E: ± AS + E: 0
PCP **Antiserum dilutions**	**1/100**	**1/200**	**1/400**	**1/800**	**1/1600**	**1/3200**	**Controls**
AS absorbed 4 ×							
AG + AS + C′	4+	$3^1/_2$+	$2^1/_2$+	1+	1+	0	AG + C′ + E: ± AG + E: 0
AS + C′	±	0	0	0	0	0	C′ + E: 0 AS + E: 0

erythrocytes as used in the test for 5, 10, 20 and 30 min respectively, in order to remove the unspecific antibodies present against human γ-globulin and the human erythrocytes used as indicator particles, the latter possibly being due to the unspecific adsorption of γ-globulin to the erythrocyte surface[13]. The low titer remaining (see Table) suggests the presence of a closely related antigen occurring in erythrocyte membranes and UCP and PCP preparations. We are presently investigating this problem. Pooled albino guinea-pig sera were used as source of complement (C′). Possibly present natural antibodies to human erythrocytes and uterine protein in guinea-pig serum were removed by absorption with human erythrocytes and lyophilized uterus homogenate. The immunediffusion experiments were performed on slides with 3.5 ml of a 1% agar gel in 0.6 *M* KCl at pH 7.4, containing 0.01% of Cialite®. The patterns developed within 4–6 days, the antigen and antiserum being refilled every second day. All IA experiments were performed at 37 °C in a shaking water bath. To 0.5 ml of a serial dilution of AS with gelatine-veronal-buffer with Ca^{++} + Mg^{++} (GVB^{++})[9] in hemagglutination tubes, 0.2 ml AG (100 μg/ml) was added and incubated for 30 min. 0.2 ml of the absorbed guinea-pig complement, (C′) diluted 1:20 with GVB^{++} was added, the samples were incubated for 60 min. 0.1 ml of EDTA-saline (0.05 *M*, pH 7.0) was added and incubated with all samples for 5 min in order to inactivate excessive complement. 0.1 ml of a 2% erythrocyte (E) suspension in EDTA-saline (type O Rh+) was added, the samples were incubated for 10 min and the allowed to settle for several hours at 37 °C or overnight at 4 °C. Readings were performed in the usual manner, graded from '0' to '4+' and were ended whenever a '2+' result had come about. Controls see Table. Rabbit serum drawn prior to imunization produced negative results.

In the Table, the results of a typical experiment are shown. Twenty μg of antigen from either uterus or placenta are sufficient to detect the antibody present in UCP antiserum dilutions of 1/600–1/1600. Preincubation of the AS with UCP lowers or abolishes the titer for this specific antigen and for PCP. This finding proves the reaction to be a specific binding of antibody and the contractile proteins. SAM gave an endpoint similar to that of the controls; it is therefore concluded that this antigen will not react with the antibody produced against UCP; these findings are in agreement with the results obtained by immunediffusion (Figure). The antigenic similarity between the UCP and PCP suggests a closer structural relationship between these proteins than was previously assumed on the basis of physico-chemical data. Admixture of other antigenic material in the placental preparations may account for the lower titer obtained in IA (Table) and possibly also for the difference in biochemical properties[2,14].

Zusammenfassung. Mit der Methode der Immunadhärenz konnte ein halbquantitativer Nachweis erbracht werden, dass Antiserum gegen kontraktiles Protein aus menschlichem Uterus mit dem homologen Antigen und kontraktilem Protein aus Placenta, nicht aber mit Aktomyosin aus quergestreifter Muskulatur reagiert.

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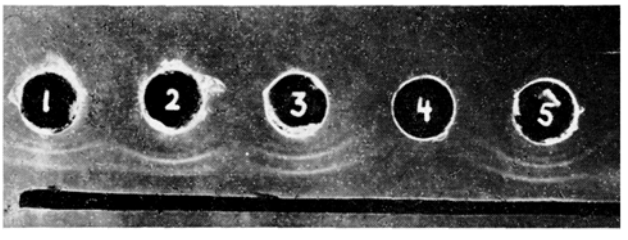

Immunediffusion test. Trough: antiserum against UCP; wells: 1, 3 + 5 = UCP, 2 = PCP, 4 = SAM.

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