happens to be comparable to the efficiency reported by ASKONAS and RHODES[8] for rat peritoneal exudate cells phagocytosing iodinated haemocyanin, 3 days following beef heart infusion broth i.p.

Had the unresponsiveness of strain XIII guinea-pigs to an antigen such as DNP-PLL been due to an inability to phagocytize it, a qualitative difference might have been expected in the cellular absorbance of ^{14}C-DNP$_{12}$PLL$_{398}$. As this was not the case, the strain XIII block inhibiting the synthesis of anti DNP-PLL antibodies is evidently 'extra-phagocytic'. It is, of course, possible to reason that a difference has been obscured in the phagocytic activity of a small, but significant class of cells. With regard to this possibility, the results demonstrate that such a class of cells, if it existed, represents less than 10% of all phagocytic cells present. A group of cells constituting a significantly greater fraction than this, would have fallen within the range of detectability. Phagocytosis of DNP-PLL at similar rates by both strains, clearly does not exclude the existence of a metabolic block in strain XIII macrophages that could inhibit subsequent processing of the antigen.

Résumé. Respectivement, les cochons d'Inde des races II et XIII immunisés répondent et ne répondent pas au poly-L-lysine conjugués. Par contre, nous savons qu'ils sont également capables de phagocyter le ^{14}C-DNP-PLL dans la mesure où le sont les cellules des exudats du péritoine.

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The Role of the Thymus in Antinuclear Autoimmunization

In earlier experiments[1–5] it was shown that thymectomy of the new-born mouse leads to the appearance of antinuclear antibodies in a high percentage of cases. Other authors have also noted the appearance of autoimmunization symptoms after thymectomy, in mouse and rabbit[1,6–8]. To explain this autoimmunization two principal theories were advanced: (1) Thymectomy might be directly responsible for the formation of autoantibodies if it is recognized that one of the functions of this organ is inhibition of development of autoimmune reactions. (2) Auto-immunization might also be an indirect result of suppression of the thymus via the immune defect following thymectomy, which permits invasion of the organism by pathogens (viruses especially) that might be the essential cause of the signs of autoimmunization. To obtain arguments in favour of one of these theories, a study was made of the appearance of antinuclear factors in 29 CF1 inbred mice thymectomized at the age of 1 month. The antibodies were determined by the immunofluorescence reaction on blood smears from mice infected with trypanosoma gambiense according to usual technique[9]. The animals subjected to late thymectomy did not show any immune defect for several months, but from the third months onwards (Table I) antinuclear antibodies were seen to develop in a manner analogous to that seen in the CF1 mice thymectomized at birth. Nevertheless, the percentage of positive reactions in the animals thymectomized at the age of 1 month was lower than that found in the CF1 mice thymectomized at birth: 50% as against 71%. These findings appear to favour hypothesis (1), direct regulation of the autoimmunity by the thymus. The animals did not have the immune defect seen in the animals thymectomized at birth and invasion of a pathogen cannot be used to explain the autoimmunization. The smallest percentage of animals with autoantibodies in this experiment, compared with the animals thymectomized at birth, should be compared with the results obtained in the pathogen-free CF1 mice thymectomized at birth[2], where positive results amounted to 25% only. It seems, therefore, that although an infection is not necessary for the appearance of antinuclear antibodies, it may be an accessory favouring factor.

Again to permit a choice between the two principal hypotheses proposed, a study was made of the possibility of adoptive transfer of the antinuclear autoimmunity appearing in the animals thymectomized at birth. This was done in the following manner.

Table I. Antinuclear antibodies in CF1 mice thymectomized at the age of 1 month

Titres	Number of months after thymectomy						
	3	4	5	6	7	8	9
8	4	6	5	5	5	6	5
64	–	–	6	6	6	4	4
512	–	–	1	1	1	2	2
Total of mice with antinuclear antibodies	4	6	12	12	12	12	11
Total number of mice	29	27	26	25	25	24	22
% of animals with antinuclear antibodies	13.8	22.2	46.1	48	48	50	50

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Table II. Adoptive transfer of antinuclear autoimmunity

Antinuclear antibody titre of the donors	8		64		512		1024		1024		1024		1024	
Recipients 1 month old thymectomized at birth	3	4	1	2	5	6	7	8	9	10	11	12	13	14
No. of days after transfer														
8	8	4	2	0	8	8	4	2	4	8	0	8	2	0
15	64	8	2	0	8	64	64	2	8	8	dead	64	2	0
25	2058	8	0	0	8	dead	64	0	4	8		64	0	0
55	4096	8	0	0	dead		64	dead	dead	dead		dead	dead	0
75	4096	0	0	0			dead							0

The spleen was collected from a CF1 animal thymectomized at birth, in which antinuclear antibodies had appeared at the end of several months. The spleen was cut into 4 fragments of equal size. Each of these fragments was implanted into the peritoneal cavity of 1-month-old CF1 mice. Two of these recipient animals were normal and served as controls, the other 2 were mice thymectomized at birth. The antinuclear antibodies were determined in the recipients at the end of 8 days, and then periodically as indicated in Table II. Seven CF1 mice thymectomized at birth with antinuclear antibodies served as donors. 28 mice of 1 month of age, 14 of them thymectomized at birth, were used as recipients.

It is seen (Table II) that 11 of the recipient animals thymectomized at birth had antinuclear antibodies from the 8th day of implantation onwards, sometimes at high titres, whereas none of the 14 controls showed a positive reaction. Adoptive transfer of antinuclear autoimmunization was therefore possible only in animals thymectomized at birth. How can these results be interpreted? The antibodies found in the recipient mice will originate from adoptive transfer and not from autoimmunization of the recipient, which in thymectomized animals can develop only after the third month. It seems that transferred spleen cells can multiply or continue to secrete antinuclear antibodies only if the recipient is thymectomized. Thymus seems to have an inhibiting effect on the autoimmune reactions. Another possibility is that the grafted cells multiply easily in the thymectomized animals by occupying depopulated thymus-dependent zones in the animals thymectomized at birth. This possibility cannot be rejected, but the results of late thymectomy of the CF1 mice combined with those of adoptive transfer experiments appear to favour the hypothesis of a regulatory effect of the thymus on autoimmune reactions.

The high number of deaths among the 14 recipient thymectomized animals may seem surprising and lead us to think that these mice died from a graft-versus-host reaction in relation to a minimum antigen difference in the inbred CF1. In fact, the percentage of deaths among those animals is the same as that we obtained in a previous study[4] with CF1 thymectomized at birth.

Recipient animals died with symptoms of wasting disease, which is not surprising since these mice had been thymectomized at birth and the spleen cells, which had been transfered to them, were of not use to prevent the wasting disease since these cells came from animals thymectomized at birth[10].

Résumé. La thymectomie chez des souris CF1 âgées de 1 mois est suivie comme dans le cas de la thymectomie néonatale de l'apparition d'anticorps antinucléaires. Le transfert adoptif de l'autoimmunité antinucléaire apparu chez des animaux thymectomisés à la naissance n'est possible que si les animaux receveurs sont eux-mêmes thymectomisés. Ces constatations mettent en évidence un rôle possible de contrôle des réactions autoimmunes par le thymus.

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[10] With the technical cooperation of Mrs. D. TILLE.

Comparative Effects of Sulfhydryl Inhibitors on Melanosome Movements within Vertebrate Melanophores

Sulfhydryl groups have been implicated in the regulation of both melanosome (melanin granule) movements[1,2] and the process of melanogenesis[3,4]. The sulfhydryl inhibitors mersalyl[2,5] and N-ethyl maleimide[2] cause in vitro darkening of the skin of the lizard[2] and the frog[5] by dispersing melanosomes within dermal melanophores. Other sulfhydryl inhibitors such as parachloromercuribenzoate, iodoacetamide, and mercuhydrin also are said to darken frog skins and, in addition, are said to inhibit MSH activity[1]. Because these agents appear to mimic the action of melanophore-stimulating hormone (MSH) on these pigment cell organelles it has been suggested that sulfhydryl groups may play an important role in the mechanism of action of MSH[2]. We have further investigated the effects of sulfhydryl inhibition on both melanosome dispersion and aggregation. In the present communication we compare the effects of a number of closely related sulfhydryl inhibitors and clarify the structural requirements necessary for such inhibition.

Methods and materials. The effects of sulfhydryl inhibitors on melanophore responses of the frog, *Rana pipiens*, and the lizard, *Anolis carolinensis*, were studied,