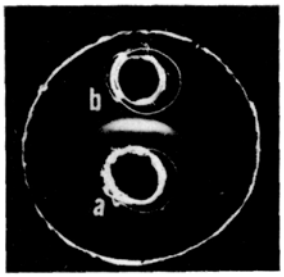
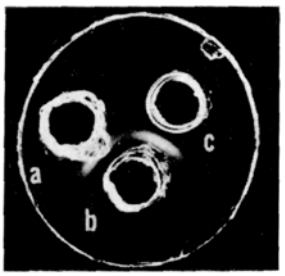
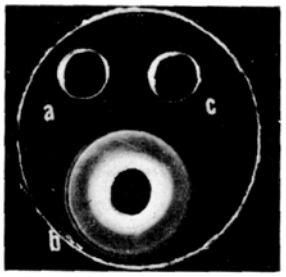


1



2



3

Photographs of Ouchterlony plates (after 18 day course of immunization). – Fig. 1. Depot a, containing roach-protozoa antigen; b, anti-roach rabbit serum. – Fig. 2. Same as in Figure 1, only c depot added containing termite-protozoa antigen. – Fig. 3. Control.

known termites, because many of the protozoa of termites have not been adequately identified (CLEVELAND[10]). Thus, some other means must be employed to attempt to show this relationship (if there exists such a relationship).

This study was undertaken in an attempt to establish a method whereby immunological responses between protozoa symbiotic to a roach and a termite might be employed to provide chemical evidence to support existing morphological evidence for protozoan relationship in the dissimilar hosts.

As part of this investigation, animal sensitization to hind-gut protozoa of the roach *Cryptocercus* was conducted in rabbits according to 'standard immunological protocol'. Anti-roach rabbit serum resulted in interfacial ring and flocculation tests indicating an antibody titer of 1/1024. Results of agar diffusion according to Ouchterlony (Figures 1–3) indicated a similar antibody response between protozoa symbiotic to the roach *Cryptocercus* and the termite *Zootermopsis*. Subsequent agglutination tests revealed individual clumping of two different genera of protozoa (*Trichonympha* and *Monocercomonoides*) of which *Trichonympha* is common to both roach and termite. Results at present indicate close chemical affinity between protozoa of the roach *Cryptocercus punctulatus* and the termite *Zootermopsis nevadensis*[13].

Résumé. L'analyse des responsabilités immunologiques après immunisation d'animaux expérimentaux avec les protozoaires symbiotiques du 'roach', *Cryptocercus* et du termite *Zootermopsis* indique une étroite affinité chimique entre les protozoas de ces hôtes. Les résultats semblent confirmer indirectement la taxonomie morphologique courante des hôtes.

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[13] Grateful acknowledgment is made to Dr. H. RITTER JR. for guidance during the course of this investigation.

Aspermatogenic Antigen from Brain

It has been known for many years that brain and testicle share some antigenic similarities in that antisera to these two organs cross-react[1]. The nature of the common antigen(s) is unknown. It has been reported that homologous brain homogenate can induce testicular damage in guinea-pigs[2]. Since homologous testicular homogenate also causes testicular lesions, the inference is that antigenic similarity of the brain and testicle includes an aspermatogenic factor (ASF). However, inasmuch as injection of brain homogenate can induce acute disseminating encephalomyelitis, the testicular dyscrasia observed in such animals could result from a general debilitating condition. The current experiments were designed to determine the immunologic specificity of the brain antigen causing testicular damage.

The experimental design is indicated in the Table. The general procedure for preparing the adjuvant-antigen emulsions and the injection routine have been described in detail elsewhere[3,4]. The extraction methods that have been applied to testes[5] were also applied to brain. Brains were obtained from 50 exsanguinated male and 50 exsanguinated female guinea-pigs. The brains were kept at – 20°C until used. For further purification of the extracted antigens, modifications of the extraction were employed: these included re-precipitation with trichloracetic acid, extensive dialysis against running tap water (72 h), and partitioning between chloroform and butanol using the aqueous phase which was lyophilized after further dialysis.

From the Table, it can be seen that brain extracts were potent in inducing depletion of spermatogenic tissue as indicated by the testicular damage rating in which increasing numerals (+ 1 to + 4) denote increased damage[4]. A curious finding which requires further work for substantiation was that extract of female brain was more potent in inducing hypospermatogenesis or aspermatogenesis than was the extract of male brain.

In view of these results in which testicular damage was elicited by brain extracts without accompanying neuro-

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Testicular damage in guinea-pigs injected with homologous brain and testicular homogenates and extracts[a]

No. of animals	Material injected[b] and amount (mg) per animal	Average paired weight (g) of testes (range)	Testis damage rating				
			0	+ 1	+ 2	+ 3	+ 4
8	Adjuvant and saline	4.6 (3.6–5.0)	8				
8	Brain homogenate (250 mg)	2.5 (1.5–3.8)	2	2	2	2	
8	Testicle homogenate (250 mg)	1.2 (0.9–1.5)				1	7
11	Brain extract (10 mg)	2.5 (1.0–4.6)	3	3	2	1	2
7	Male brain extract (10 mg)	3.1 (2.0–4.6)	3	1	3		
4	Female brain extract (10 mg)	1.5 (1.0–2.1)			1	1	2
2	Re-extracted brain (10 mg)	1.0 (1.0–1.1)					2
8	Testicle extract (10 mg)	1.0 (0.9–1.3)					8

[a] Duration of experiment = 2 months. [b] In Freund's adjuvant.

ogic symptoms, it appears that the extracts were free of encephalitogenic antigen. We therefore made comparisons of carbohydrate, protein, and amino acid content of the brain extracts and of the testicular extracts using the DREYWOOD[6], the LOWRY[7], and SPACKMAN, STEIN, and MOORE[8] procedures, respectively. Brain antigen contained 700–900 μg/mg of protein (using bovine albumin as standard) and 15–35 μg/mg of carbohydrate (using glucose as standard). These data plus the amino acid analyses, when compared with those obtained for testicular ASF[9,10] show striking similarities. The subtle differences between the two antigens are being explored currently. At this time, however, it can be said that brain and testicle share common antigenicity, which includes an antispermatogenic factor[11].

Zusammenfassung. Antigenetisches Material aus Meerschweinchenhirn extrahiert, rief, wenn es Meerschweinchen injiziert wurde, Aspermatogenese hervor. Hirnantigen verglichen mit demjenigen aus homologen Testikeln liess zahlreiche Ähnlichkeiten in Eiweiss-, Kohlehydrat- und Aminosäurengehalt erkennen. Hirn und Testikel besitzen somit gemeinsame Antigenität (welche das Aspermatogenese-Antigen einschliesst), und das Aspermatogenese-Antigen kann vom encephalitogenen Faktor abgetrennt werden.

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Beobachtung über den Einfluss der Laktation auf die Erhaltung der Blastocysten bei kastrierten Mäusen

Bei den Muriden wird, wenn das Weibchen beim postpartum-Östrus befruchtet worden ist und den vorhergehenden Wurf von mehr als zwei Jungen säugt, die Implantation der befruchteten Eier bis zum Abklingen der Laktation verzögert, so dass die Tragzeit von 3 bis auf 5 Wochen verlängert werden kann. Während der Latenzzeit bleiben die Blastocysten frei im Uteruslumen suspendiert. Das Unterbleiben der Nidation ist eine Folge der Laktation; werden der säugenden Mutter die Jungen weggenommen, so erfolgt die Nidation innerhalb von 2–4 Tagen[1]. Nach unserer Annahme ist die Verzögerung die Folge eines Östrogenmangels; man kann bei säugenden Weibchen durch Zufuhr einer kleinen Östrogenmenge die Implantation rechtzeitig oder in einem beliebigen Zeitpunkt der Laktation herbeiführen. Bei Weibchen, die nach der Befruchtung kastriert worden sind, kann man mit Progesteron die Keime am Leben erhalten und dann mit Östrogen die Nidation provozieren. Welches der Faktor ist, der beim säugenden Weibchen die Östrogenausschüttung unterbindet, ist nicht bekannt.

Wir haben uns nun gefragt, ob ein bestimmtes Agens beim säugenden Weibchen die im Uterus suspendierten Keime am Leben erhält und ob man während der Laktation beim kastrierten Weibchen auch ohne Progesteron die Blastocysten erhalten und später zur Nidation bringen könnte. Ratte und Maus gehören zu den Tierarten, bei denen die Kastration in jedem Zeitpunkt die Gravidität unterbricht[2]. Als einzige Ausnahmen von dieser Beobachtung kennen wir die Mitteilung von COURRIER und COLONGE[3], wonach bei der Ratte nach der am 12. Tage

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