

Zeitschrift: Acta Tropica
Herausgeber: Schweizerisches Tropeninstitut (Basel)
Band: 26 (1969)
Heft: 2

Artikel: Immunoglobulins and antibodies in "Borrelia turicatae" infections
Autor: Felsenfeld, Oscar / Wolf, Robert H.
DOI: <https://doi.org/10.5169/seals-311613>

Nutzungsbedingungen

Die ETH-Bibliothek ist die Anbieterin der digitalisierten Zeitschriften auf E-Periodica. Sie besitzt keine Urheberrechte an den Zeitschriften und ist nicht verantwortlich für deren Inhalte. Die Rechte liegen in der Regel bei den Herausgebern beziehungsweise den externen Rechteinhabern. Das Veröffentlichen von Bildern in Print- und Online-Publikationen sowie auf Social Media-Kanälen oder Webseiten ist nur mit vorheriger Genehmigung der Rechteinhaber erlaubt. [Mehr erfahren](#)

Conditions d'utilisation

L'ETH Library est le fournisseur des revues numérisées. Elle ne détient aucun droit d'auteur sur les revues et n'est pas responsable de leur contenu. En règle générale, les droits sont détenus par les éditeurs ou les détenteurs de droits externes. La reproduction d'images dans des publications imprimées ou en ligne ainsi que sur des canaux de médias sociaux ou des sites web n'est autorisée qu'avec l'accord préalable des détenteurs des droits. [En savoir plus](#)

Terms of use

The ETH Library is the provider of the digitised journals. It does not own any copyrights to the journals and is not responsible for their content. The rights usually lie with the publishers or the external rights holders. Publishing images in print and online publications, as well as on social media channels or websites, is only permitted with the prior consent of the rights holders. [Find out more](#)

Download PDF: 20.08.2025

ETH-Bibliothek Zürich, E-Periodica, <https://www.e-periodica.ch>

From the Tulane University Delta Regional Primate Research Center,
Covington, Louisiana, 70433

Immunoglobulins and Antibodies in *Borrelia turicatae* Infections*

OSCAR FELSENFELD and ROBERT H. WOLF

The relationship of immunoglobulins (Ig-s) and immunocytes (immunologically active cells, IAC) to antibodies against *Borrelia* has not yet been studied, and no comparison of blood and lymphatic tissue Ig levels had been made in this infection. The immunological variations and mutations of the infecting *Borrelia* strains during relapses [reviewed by GEIGY & HERBIG (1955), FELSENFELD (1965) and BURROWS (1968)] make the exploration of these factors during and after episodes of borrelemia of special interest. Therefore, this communication reports the results of experiments in which Ig-s and IAC were studied and related to circulating and lymph node antibody levels in patas monkeys (*Erythrocebus patas*) artificially infected with *Borrelia turicatae*.

Materials and Methods

Two male patas monkeys (*Erythrocebus patas*), weighing 4.5 and 5 kg, respectively, were each infected with intramuscular injections of 60 to 70 *Borrelia turicatae* isolated from *Ornithorodos turicata* ticks. The monkeys were considered to be free of disease by routine clinical, microbiological, and chemical examinations made prior to the infection. During the experiment, the animals were kept in strict isolation and handled only by caretakers who were free from respiratory, skin and intestinal infections.

The ticks from which the borreliae were isolated were obtained through the kindness of Dr. W. Burgdorfer, U.S.P.H.S. Laboratories, Hamilton, Mont.

Heparinized blood samples were collected from each animal before infection, twice a week during the first five weeks after infection, and again two months after infection. Borreliae (if present) were recovered from the blood samples by centrifugation at 1,500 g for 30 min. The plasma was used for antibody studies. Sterile distilled water was added to the sediment, and the laked blood centrifuged at 12,000 g and 2 to 4°C. Additional borreliae became available from rats inoculated with the blood of infected monkeys.

Peripheral lymph nodes were extirpated aseptically from each animal, while it was anesthetized with Sernylan® (Parke, Davis and Co.) on days 3, 14, and 35 after infection. The lymph nodes were divided into two parts, one of which was frozen immediately in N₂ to be used in the preparation of cryostat sections for fluorescent microscopy, and for Ig determinations. The other half of the specimen was fixed in neutral buffered formalin, embedded in paraffin, sectioned, and stained with hematoxylin and eosin or with methyl green and pyronin.

Ig determinations were performed by mixing part of the tissue sample with an equal amount (w/v) of 0.01 M phosphate buffer containing 0.16 M NaCl,

* The experiments reported herein were conducted according to the "Principles of Laboratory Animal Care" of the U.S. National Society for Medical Research.

pH 7.2 (BPS), and disintegrating it by ultrasonic vibration in the Sonifer apparatus (Branson Instruments Co.) at 20 Kc/sec, 100 W and 5 A output, for 5 minutes under constant cooling to -5°C .

The debris was centrifuged off at 12,000 g and 2 to 4°C , and the supernatant examined for Ig content.

Ig levels were determined in plasma and tissue supernates by the single cell radial diffusion method of MANCINI et al. (1965) using rabbit anti-patas IgG, IgA and IgM sera prepared in this laboratory, by injecting pairs of adult rabbits with 20 mg of the respective Ig in Freund's complete adjuvant intracutaneously, and after one month, 4 times in weekly intervals, with 5 mg Ig intravenously. The patas Ig-s were separated by the procedure of VAERMAN et al. (1963). The rabbit sera were tested by agar gel electrophoresis and in the agar double diffusion test against whole patas serum as well as against separated Ig-s for specificity. Only those giving one single specific line were used in these (and other) experiments (FELSENFELD et al., 1968). Anti-IgD serum was employed in tissue studies. It was obtained through the courtesy of Dr. David Rowe, Director of the W.H.O. Immunoglobulin Reference Laboratories, Lausanne, Switzerland. This serum was an anti-human IgD serum but it reacted with patas IgD to 72 ± 8 per cent of its titer.

All sera were kept at -20°C without a preservative.

Immobilizine titers were determined by a method similar to that of VAISMAN & HAMELIN (1954) by mixing 0.1 ml of two-fold serial dilutions of heat-inactivated (56°C for 30 min) plasma and tissue supernatants in BPS with equal volumes of a suspension of *Borrelia* in BPS containing $2,000 \pm 15$ organisms per ml. Controls containing only borreliae and BPS were also set up. The mixtures were incubated in a moist chamber for 30 min and examined under the phase microscope. The number of immobilized organisms was counted and compared with the number of non-motile borreliae in the controls. The dilution causing immobilization of $50 \pm 5\%$ of the organisms was considered the 50% immobilizine titer (I_{50}).

Borreliolysin was determined by a modification of the procedure used by BALTEANU et al. (1948), adding 0.02 ml of 1:20 diluted guinea pig complement to aliquots of plasma and tissue supernatants prepared as for immobilizine determinations. The number of intact borreliae in the tissue and plasma dilutions was counted under the phase microscope and the 50% borreliolysin (B_{50}) titer calculated as in the determination of the 50% immobilizine titer by comparison with the controls. Complement alone, in the dilution used in these experiments, had no significant borreliolytic activity.

Antigen was demonstrated in frozen sections of lymph nodes prepared in the Harris cryotome at -15°C by fluorescent microscopy. The sections were dried, fixed with 10% buffered formalin, and washed in BPS. A Leitz Ortholux microscope with Osram bulb HBO 200, and BG 38 and UVI filters, were used. Rabbit anti-*B. turicatae* serum (prepared by the senior author during his work at the Walter Reed Army Medical Center in Washington, D.C.) followed by application of goat anti-rabbit fluorescein-labeled serum were employed.

The *enumeration of IAC* producing the respective Ig-s was made using the same sandwich technique. The rabbit anti-IgG, IgA, IgM, and IgD sera employed in Ig determinations in fluids (see above) were used, followed by fluorescein-conjugated goat anti-rabbit serum. The enumeration of the cells forming Ig-s of different classes was performed according to the technics recommended by CRABBÉ et al. (1965) and CRABBÉ & HEREMANS (1966). After the application of the specific anti-Ig serum and the fluorescein-labeled antiserum, the number of cells showing fluorescence were counted over a delineated area. The total

number of potential IAC was enumerated in a parallel section stained with methyl green and pyronin. (It is also possible to wash the same section several times with BPS, fix again in Carnoy's fluid, and stain with the Unna-Pappenheim method.)

Separation of Ig-s from plasma was done by a modification of the classical method of VAERMAN et al. (1963) (FELSENFELD et al., 1968). Gel filtration of the Ig-containing material through Sephadex G-200 and collection of fractions after elution with 0.2 M Tris buffer pH 8 was the first step. Each protein-rich fraction (monitored by a Gilson UV meter at 280 m μ) was tested for Ig content by the method of MANCINI et al. (1965) against the specific anti-Ig sera, then chromatographed on DEAE Sephadex, using phosphate-saline buffer with increasing molarity (0.1 to 0.3) and decreasing pH (7.6 to 6.6). The fractions were concentrated by evaporation under a fan in the cold room, followed by dialysis against polyvinyl pyrrolidone. Purity was controlled by double gel diffusion and agar gel electrophoresis against whole anti-patas antiserum, and the specific anti-Ig sera. The solutions were standardized to contain 10 mg protein per ml with the aid of the biuret test, and kept at -20°C .

Avidity test. The antigen for the experiments with antibody-antigen complexes was prepared by suspending 1 to 1.3×10^6 borreliae from rat blood in 1 ml BPS, disintegrating them in the Sonifer apparatus at 20 Kc/sec 120 W 6 A and -5°C for 5 minutes and centrifuging the suspension four times at 12,000 g and 2 to 4°C . The sediment was washed with 1 ml BPS after each centrifugation. The supernates and the washings were pooled, evaporated under a fan in the cold room, and concentrated by dialysis against polyvinyl pyrrolidone. Preliminary tests showed that optimal precipitation with 10 mg% Ig solutions was obtained by adding 0.2 to 0.4 ml of this antigen to 1 ml Ig solution, according to the sub-class of the Ig. The precipitation was carried out by mixing antibody and antigen, allowing the mixture to stand at room temperature for 2 h, then overnight at 2 to 4°C . The sediment was centrifuged three times at 12,000 g and washed each time with 1 ml BPS. Finally, the sediment was centrifuged at 15,000 g at 2 to 4°C and the supernate drained off in the cold room. Eight tubes were prepared with each antibody (Ig)-antigen mixture.

For the determination of the avidity (affinity) of the antibody, 4 ml aliquots of buffer solutions with pH increasing stepwise by 0.5, were added to the series of 8 tubes containing antibody-antigen precipitates. Glycine hydrochloride-glycine buffer was employed at pH 3.0 and 3.5, and acetate-acetic acid buffer at pH 4.0 to 6.0. A control tube contained only the optimal amount of the antibody (Ig). All tubes were shaken at room temperature for 30 min, then chromatographed through a CM cellulose column, using 0.05 M sodium hydrocarbonate as the eluent. The effluents containing Ig were pooled, and concentrated by pressure dialysis according to TOZER et al. (1962). The amount of Ig was determined by the MANCINI et al. (1965) method against homologous anti-Ig sera, and the per cent of dissociated Ig calculated by comparison with the control.

All tests were carried out in duplicate or triplicate and, if indicated, under sterile conditions.

Results

The animals survived the infection without overt signs and symptoms of disease. Borreliae appeared in the blood from day 7 to 14, then again on days 21 and 24 after infection, corresponding to one relatively long primary attack, and a shorter relapse. The

number of borreliae reached 4900 and 4560, respectively, on day 14, and 1660 and 2230, respectively, per 1 ml blood on day 24 in the two animals. Borreliae from both episodes in the monkeys were used for immunological studies. Rats were inoculated with borreliae from either episode but organisms appearing only during the first attack in the rat were used for preparation of antigens to avoid the employment of antigenic variants that may develop during relapses.

Fluorescent microscopy permitted the visualization of *Borrelia* antigen in the lymph nodes on days 3 and 14 but not on day 35. Fluorescence was observed in a number of cells classified as phagocytic elements but not in immature lymphoid-plasma cells.

An increased number of eosinophilic cells was seen in one of the monkeys in the sinuses of the lymph nodes on days 14 and 35. The number of germinal centers increased between day 3 to days 14 and 35, and the number of pyroninophilic cells also became greater in both animals. Endothelial and phagocytic cells appeared in the sinuses in increased numbers. Fluorescent microscopy revealed that the Ig-forming cells were localized predominantly at the periphery of the lymphoid nodules and along lymphatic cell strands, although some appeared also in efferent lymph pathways. On day 35, the germinal centers contained considerable amounts of fluorescent material between the cells.

Table 1 demonstrates the relationship of borrelemia, serum Ig-s, immobilizine and borreliolysin titers in the two experimental animals. The period of days 7 to 14 was designated that of the primary (initial) attack; days 21 to 24 the relapse. Serum IgA varied between 189 and 210 in the first, and 205 and 228 in the second patas, without apparent relationship to the course of the infection. Its levels are not shown in the table. IgG followed an ascending course, with remissions at the ends of the borrelemiae. IgG levels were still elevated 60 days after the infection (36 days after the second borrelemia). IgM also increased until the 28th day of the disease but decreased during the episodes of borrelemia. The first elevation of IgM was seen on day 3, whereas IgG was not found in larger amounts until day 10.

Immobilizine titers higher than 1 : 16 appeared in one patas at the end of the primary attack but only after the initial attack in the other patas against borreliae isolated from that episode. These titers decreased at the end of the relapse, then increased until day 60. Immobilizine against borreliae from the second attack appeared only in sera collected at the end or after the relapse. The titers at the end of the experiment were lower than those of the immobilizine against borreliae from the primary attack.

TABLE 1
Borreliae in the blood; serum Ig, immobilizine and borreliolysin titers

Day after infection	No. of Borreliae per ml		Ig in mg %		Reciprocal 50 % titers of			
					Immobilizine against		Borreliolysin against	
	P 1 ³	P 2 ³	P 1	P 2	M		G	
			P 1	P 2	Borreliae I ¹	Borreliae II ²	Borreliae I ¹	Borreliae II ²
			P 1	P 2	P 1	P 2	P 1	P 2
0	0	0	725	830	0 ⁴	0	0	0
3	0	0	756	817	0	0	0	0
7	560	1210	749	880	0	0	0	0
10	2340	3120	1057	1097	0	0	0	0
14	4950	5270	956	832	0	0	0	0
17	0	0	1113	984	64	128	32	0
21	210	440	1052	967	107	128	32	0
24	1660	2230	883	822	32	85	64	0
28	0	0	1105	1067	219	256	128	32
31	0	0	1091	1115	256	853	85	128
35	0	0	1508	1247	512	853	128	85
60	0	0	1266	1078	512	2048	256	219

Averages of 3 determinations.

¹ Borreliae from initial attack.

² Borreliae from relapse.

³ Patas 1 and patas 2, respectively.

⁴ 16 or less.

TABLE 2

Antigen, IAC¹, Ig, immobilizine and lysin titers in lymph nodes

Test	Specimen collected days after infection					
	3		14		35	
	P 1 ²	P 2	P 1	P 2	P 1	P 2
Antigen by immunofluorescence	++	++	+++	+++	0	0
IAC per 10 ⁶ cells						
Class IgG	70	87	152	149	150	141
IgA	22	18	32	54	25	20
IgM	42	59	67	81	32	27
Ig in μ g per mg tissue						
IgG	8.7	10.1	18.1	23.1	20.2	28.3
IgA	4.3	3.8	4.8	4.4	4.0	4.2
IgM	12.5	17.4	20.2	25.2	11.2	10.3
Immobilizine reciprocal titer against						
Borreliae I ³	0 ⁵	0	64	64	219	256
Borreliae II ⁴	0	0	0	0	64	174
Borreliolysin reciprocal titer against						
Borreliae I	0	0	0	32	128	219
Borreliae II	0	0	0	0	85	128

Averages of 3 determinations.

¹ Immunologically active cells.² Patas 1 and patas 2, respectively.³ Borreliae from initial attack.⁴ Borreliae from relapse.⁵ 16 or less.

Borreliolysin titers did not reach the height of the immobilizine titers. They appeared against borreliae from the initial attack in one patas immediately after the primary borrelemia, in the other animal after both episodes. Lysins against borreliae from the second episode reached levels higher than 1 : 16 after the second attack. As immobilizines, the borreliolysin titers against borreliae from the relapse were also lower than against the organisms from the primary attack.

Table 2 shows the occurrence of antigenic material, IAC, Ig-s, immobilizine, and borreliolysin in the extirpated lymph nodes.

The number of antigen-containing cells demonstrated by the immunofluorescent method is shown semi-quantitatively. IgD did not reveal statistically significant fluctuations. There were 3 to 9 IgD forming cells per 10⁶ potential immunocytes. Their exact num-

bers are not presented in the table. The proportion of Ig-forming cells of the other 3 Ig classes studied was higher than in the not infected controls (not shown in Table 2). In normal patas, the background count of IAC was less than 10 per 10^6 cells. In the infected animals, the number of IAC was higher on all days when lymph nodes were examined. The increase of IgG-producing cells was the greatest and prevailed until day 35, whereas IgM-producing IAC decreased between days 14 and 35. IgA followed the pattern of IgM. Ig production ran parallel to IAC formation to a certain degree. The relatively small fluctuations of IgA may have been due to the inherent difficulties in determining small amounts of IgA. The ratio Ig : IAC was (approximately) 1 : 8 for IgG; 1 : 4 for IgA; and 1 : 3 for IgM.

Immobilizine and borreliolysin against organisms from the primary attack appeared in the lymph nodes early, while the borrelemia still lasted. Immobilizine titers were lower than in the blood on day 35 but borreliolysin titers matched serum titers or even exceeded them.

The changes in the avidity of IgG and IgM isolated from the blood of the monkeys are shown in Table 3. Only Ig-s from sera with significant immobilizine and/or borreliolysin titers were examined. The pH at which the antibody-antigen complexes could be dissociated, decreased with time. Especially IgM-antigen com-

TABLE 3
Avidity of serum antibody produced against borreliac

Serum collected on day after infection	pH at which antibody-antigen complex dissociated							
	IgG-Borreliac I ¹		IgG-Borreliac II ²		IgM-Borreliac I		IgM-Borreliac II	
	P 1 ³	P 2	P 1	P 2	P 1	P 2	P 1	P 2
14	ND	4.25	ND	ND	ND	5.5	ND	ND
17	4.0	4.0	ND	ND	5.25	5.0	ND	ND
21	3.75	3.5	ND	ND	5.0	5.0	ND	ND
24	3.0	3.25	ND	5.0	5.25	5.0	ND	5.25
28	3.25	3.0	4.5	4.25	4.5	4.75	5.0	4.5
31	3.25	3.0	4.0	3.75	4.0	4.25	4.5	4.5
35	3.0	3.0	3.5	3.5	4.25	4.0	4.25	4.5
60	3.0	3.0	3.25	3.5	3.25	3.5	3.25	3.75

Averages of 2 determinations.

ND = not done.

¹ Antigen from borreliac isolated from initial attack.

² Antigen from borreliac isolated from relapse.

³ Patas 1 and patas 2, respectively.

plexes were dissociated more easily during the early days after infection. At the end of the experiment, on day 60, both Ig-s showed strong avidity for *Borrelia* antigens. IgG, however, displayed strong affinity already on days 31 and 35.

Discussion

The experiments were carried out only on two monkeys. This number of experimental animals may appear small. However, when the great number of tests per animal is considered, it becomes evident that the simultaneous testing of more patas would have required an unusual number of investigators, much apparatus and many laboratory procedures, especially since control animals also had to be examined. The results show good agreement between the findings in both patas, considering that individual variations may be significant. This seems to justify the restrictions put on the number of the animals used.

The *Borrelia turicatae* strain employed in this project was of low pathogenicity. Monkeys have been found susceptible to borreliæ (reviewed in FELSENFELD, 1965). However, in these experiments clinical symptoms did not develop, and the borrelemia was of a low level. The interval between the two attacks was short. The antibody formation remained below the titers observed in man by BALTEANU et al. (1948) a.o.

The circulating (serum or plasma) Ig-s showed an early increase of IgM, followed by mounting IgG levels. After 60 days, only IgG remained elevated. IgG production replaces initial IgM responses in many infections. Prolonged IgM manufacturing in lymphoid-plasma cell tissue is a characteristic of a chronic infection or other disorder that is able to increase Ig production beyond the background level (cf. review by RITZMANN & LEVIN, 1967). It is possible that borreliæ disappear from the blood stream but are retained in certain organs, such as the central nervous system (cf. reviews by GEIGY & HERBIG, 1955; FELSENFELD, 1965; GEIGY, 1968), giving impetus to further Ig generation. SEVAG (1967) discussed the shut-off of IgM formation by a possible feedback mechanism when IgG manufacturing reaches a critical level. According to that concept, intensive IgM formation should not take place when IgG prevails, principally not when IgG with strong avidity is being produced. The feedback mechanism which cuts off IgM production does not seem to become functional in borreliosis until a late date, in our experiments sometimes around day 35. The antigenic mechanism of trypanosomes resembles that of borreliæ to a certain extent (FELSENFELD et al., 1965). It is possible that the high IgM titers

seen in African sleeping sickness (MATTERN et al., 1961) emanate from a similar impulse for antibody formation as in borreliosis.

The simultaneously increasing IgG and IgM levels are not dissimilar to those described in diseases as far apart as malaria (ABELE et al., 1965) and infectious mononucleosis (WOLLHEIM & WILLIAMS, 1966). Surveys of the literature (WALL, 1958; HEREMANS, 1960; FAHEY, 1965), however, do not report data on relapsing fever. It is believed, therefore, that the data presented in this communication are of interest, especially since another cyclic disease, malaria, shows a similar Ig response. The proliferation of IAC in the lymph nodes and the production of Ig by them followed a pattern that might not appear unique if it were examined also in other diseases.

The immobilizine and borreliolysin levels varied according to expectation. Considering the phase variations of *Borrelia* strains (SCHUHARDT, 1942; GEIGY & BURGDORFER, 1951; BURROWS, 1968), the lack of immobilizines and borreliolysins at a level of 1:32 or higher against organisms from the relapse during and after the first attack was not surprising when the immunophysiology of borreliac is taken into account.

The immunofluorescent study of the antigen in lymph nodes showed that it disappeared by day 35. This, however, does not mean necessarily that *Borrelia* antigen was absent also in the central nervous system or other organs. The antigen appeared only in phagocytic cells, not in cells forming or able to form antibody. The process of antibody production and its relationship to antigen-incorporating cells requires, therefore, further investigation.

There is no easy explanation of the eosinophilic response in one of the patas monkeys. It may or may not be an expression of hyperergy. The localization of the pyroninophilic cells, the distribution of the lymphoid-plasma cells and the cells in the sinuses of the lymph nodes did not differ from those observed in a number of other diseases. Evidently this process cannot be considered typical for borreliosis.

The avidity (affinity) of the Ig-bound antibodies increased with time. A similar phenomenon was recently demonstrated by SEVAG (1967) in poliomyelitis. While IgM-antibodies abounded in the beginning of the disease, their avidity was low (the antigen-antibody complex could be easily disrupted by physical means, as e.g. changes of the pH). Our experiments showed a similar phenomenon taking place in borreliosis. IgG-antibody appeared to form more firm complexes already early in the disease, whereas IgM-antibody did not develop a strong affinity until later when its production decreased. It might appear desirable to take this phenom-

enon in consideration when serum antibodies are evaluated in the prognosis of the disease or in epidemiological serology. Due to the large molecular size of IgM (19S), IAC-s producing it are more easily demonstrated by plaque hemolysis and similar procedures but IgM with low avidity antibody, even though present in considerable amounts, may not be as significant as the smaller (7S) but more avid IgG carrying antibody with a greater affinity to the antigen.

References

- ABELE, D. C., TOBIE, J. E., HILL, G. J., CONTACOS, P. G. & EVANS, C. B. (1965). Alterations in serum proteins and 19S antibody production during the course of induced malarial infection in man. — *Amer. J. trop. Med. Hyg.* 14, 191-197
- BALTEANU, I., RUSS, M. & VOICULESCU, M. (1948). Les propriétés spirochétolytiques du sérum de convalescent de fièvre récurrente. — *Arch. roum. Path. exp. Microbiol.* 15, 310-312
- BURROWS, W. (1968). Textbook of microbiology. 19th edition. — Philadelphia, Penna: Saunders
- CRABBÉ, P. A., CARBONARA, A. O. & HEREMANS, J. F. (1965). The human intestinal mucosa as a major source of plasma cells containing γ A immunoglobulin. — *Lab. Invest.* 14, 235-246
- CRABBÉ, P. A. & HEREMANS, J. F. (1966). Distribution of immunoglobulin-containing cells along the human gastrointestinal tract. — *Gastroenterology* 51, 305-309
- FAHEY, J. L. (1965). Antibodies and immunoglobulins. — *J. Amer. med. Ass.* 194, 225-258
- FELSENFELD, O. (1965). *Borreliae*, human relapsing fever, and parasite-vector-host relationships. — *Bact. Rev.* 29, 46-74
- FELSENFELD, O., DECKER, W. J., WOHLHEITER, J. A. & RAFYI, A. (1965). Studies in *borreliae*. II. Some immunologic, biochemical and physical properties of the antigenic components of *B. turicatae*. — *J. Immunol.* 94, 805-817
- FELSENFELD, O., GREER, W. E. & JIŘIČKA, Z. (1968). Early immunoglobulin formation and hemagglutinin and bactericidal titers in *E. patas* after administration of a weakly pathogenic *Shigella* strain. — *Lab. Invest.* 19, 146-152
- GEIGY, R. (1968). Relapsing fevers. In: *Infectious Blood Diseases of Man and Animals*. Ed. by D. Weinman & M. Ristic. Vol. 2, pp. 175-216. — New York: Academic Press
- GEIGY, R. & BURGDORFER, W. (1951). Unterschiedliches Verhalten verschiedener Stämme von *Sp. duttoni* in der weißen Maus. — *Acta trop.* 8, 151-154
- GEIGY, R. & HERBIG, A. (1955). Erreger und Überträger tropischer Krankheiten. — *Acta trop.*, Suppl. No. 6
- HEREMANS, J. (1960). Les globulines sériques du système gamma. — Bruxelles, Belgium: Ed. Arscia S.A.
- MATTERN, P., MASSEYEFF, R., MICHEL, R. & PERETTI, P. (1961). Etude immuno-chimique de la B2 macroglobuline des sérums de malades atteints de trypanosomiase africaine à *T. gambiense*. — *Ann. Inst. Pasteur* 101, 382-388
- MANCINI, C., CARBONARA, A. O. & HEREMANS, J. (1965). Immunological quantitation of antigens by single cell radial diffusion. — *Immunochemistry* 2, 235-254

- RITZMANN, S. E. & LEVIN, W. C. (1967). Polyclonal and monoclonal gammopathies. — Lab. Synopsis 2, 9-50
- SCHUHARDT, V. T. (1942). Serology of the relapsing phenomenon in relapsing fever. In: Sympos. Relaps. Fever in the Americas. — Amer. Ass. Adv. Sci. Monogr. No. 18. Washington, D.C. 58-66
- SEVAG, E.-E. (1967). Diversity of antibodies formed against viruses. In: Cinadar, B (Editor): Antibodies to Biologically Active Molecules, pp. 301-348. — Oxford and New York: Pergamon Press
- TOZER, B. T., CAMMACK, K. A. & SMITH, H. (1962). Separation of antigens by immunological specificity. — Biochem. J. 84, 80-93
- VAERMAN, J.-P., HEREMANS, J. F. & VAERMAN, C. J. (1963). Studies of the immune globulins of human sera. — J. Immunol. 91, 7-10
- VAISMAN, A. & HAMELIN, H. (1954). Immunité et spécificité des immobilisines récurrentielles entre *B. duttoni* et *B. hispanica*. — Ann. Inst. Pasteur 86, 107-109
- WALL, R. L. (1958). Use of serum protein electrophoresis in clinical medicine. — Arch. intern. Med. 102, 618-658
- WOLLHEIM, F. A. & WILLIAMS, R. C. Jr. (1966). Studies on the macroglobulins of human serum. — New Engl. J. Med. 274, 61-67

Zusammenfassung

Immunoglobuline IgG, IgA und IgM wurden im Serum und in den Lymphknoten zweier mit *B. turicatae* infizierter Patas-Affen bestimmt. Es wurde die Anzahl immunologisch aktiver Zellen in den Lymphknoten geschätzt. Immobilizin- und Borreliolysin-Titer wurden zueinander in Beziehung gesetzt. Auch die Antikörperaktivität wurde bestimmt. Die Konzentration von IgM und IgG sowie die Avidität der Antikörper auf *Borrelia*-Antigen nahmen nach der Infektion zu. Folgerungen aus diesen Resultaten wurden diskutiert.

Résumé

Des immunoglobulines IgG, IgA et IgM furent déterminées dans les sérums et les ganglions lymphatiques de deux singes patas infectés de *B. turicatae*. Le nombre des cellules immunologiquement actives dans les ganglions lymphatiques a été estimé.

Les titres d'immobilisine et de borreliolysine ont été mis en relation. On a également effectué la détermination de l'avidité des anticorps. Le niveau des IgM et des IgG, ainsi que l'avidité des anticorps pour l'antigène *Borrelia* augmentent après l'infection. Les significations de ces trouvailles sont discutées.