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Autoimmunity in Trypanosome Infections
IV. Natural Infections in Hartebeest, Hyaena and Lion
in the Serengeti National Park, Tanzania

A. R. MACKENZIE * and P. F. L. BOREHAM

Abstract

Serum samples from 5 hartebeests, 10 hyaenas and 8 lions were examined for the presence of anti-liver and anti-Wassermann autoantibodies. No evidence of autoantibodies was found in hyaenas and lions. Two hartebeests showed low levels of both autoantibodies and two others of only one of the autoantibodies. Since multiple parasitic infections were found in four of the hartebeests and suspected in the other it is not possible to say which if any of the parasites is involved.

Introduction

Autoantibodies are known to be present in a variety of parasitic diseases including malaria (Davies 1944), coccidiosis (Asherson & Rose 1963), leprosy (Wager 1969) and schistosomiasis (Bassily et al. 1973). Recent studies in trypanosomiasis have shown the presence of a number of autoantibodies in rabbits, cattle and man. These include an anti-liver autoantibody, anti-Wassermann autoantibody and an autoantibody against a component of the fibrinogen/fibrin system (Mackenzie et al. 1973; Mackenzie & Boreham 1974; Boreham & Facer 1974).

Since a number of serum samples were available from naturally infected wild animals it was decided to examine these for the presence of autoantibodies as little is known about the immune response to trypanosomes in reservoir hosts.

Material and Methods

Collection of samples

Serum samples were collected from 5 hartebeests (Alcelaphus buselaphus), 10 hyaenas (Crocuta crocuta) and 8 lions (Panthera leo) in the Serengeti National Park during the 1971 trypanosomiasis survey organised by Professor Geigy (Geigy et al. 1973).

Animals were captured as described by Geigy & Kauffmann (1973). Blood samples were removed from the jugular veins of hartebeests and hyaenas and from the vena saphena of the lions. The blood was allowed to clot at ambient temperature, stored on ice until the serum could be separated by centrifugation and the serum maintained at -20 °C until tested.

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Anti-liver autoantibody

The complement fixation test (CFT) was used to measure the amount of anti-liver autoantibody present in the serum (Mackenzie & Boreham 1974). Saline extracts of ox, hyaena and cat livers were used as antigens respectively in tests for hartebeest, hyaena and lion autoantibodies.

Anti-Wassermann autoantibody

A CFT was employed using cardiolipin (Wellcome Reagents Ltd.) as antigen for all three species (Mackenzie & Boreham 1974).

Results

The table shows the results of the parasitological studies made by Professor Geigy and his colleagues (Geigy & Kauffmann 1973) and the autoantibody measurements.

All the animals examined were infected with piroplasms, probably Babesia sp. in lions and hyenas and Theileria sp. in hartebeests. All the lions and hyenas were infected with Hepatozoon sp. and in addition all the hyenas and four of the five hartebeests had microfilariae present in their blood. Trypanosomes were also common in these animals: 2 hartebeest were infected with Trypanosoma brucei and one with T. congolense. T. brucei was found in 3 hyenas, T. congolense in two hyenas and two hyenas had mixed infections. One hyaena No. 250 had trypanosomes present in the blood but it was not possible to identify the species because of damage during preparation of the slide. Six lions were infected with T. brucei and one with T. congolense. The uninfected lion No. 280 was a young lion not more than 8 months of age. No investigation of gastrointestinal helminths was made.

There appears to be no rise in either anti-liver or anti-Wassermann autoantibodies in hyaenas or lions except for hyaena 294 which showed a small elevation of anti-Wassermann antibody levels. This hyaena was not infected with trypanosomes.

Two hartebeests (253 and 258) showed slight increases in both anti-liver and anti-Wassermann autoantibodies while 257 had increased anti-liver autoantibody and 259 anti-Wassermann autoantibody only. Hartebeest No. 255 showed no increase in either of these autoantibodies.

Discussion

The results of these studies indicate that in lions and hyenas the natural parasite infections do not elicit the formation of autoantibodies as is seen in trypanosomiasis of rabbits, cattle and man (Mackenzie et al. 1973).

In contrast it appears that a proportion of the hartebeests develop autoantibodies but it is not possible to suggest which of the multiple parasite infections is responsible. It is possible that helminth infections were involved but since these animals were only immobilised for the removal of blood samples and not killed, it was not possible to investigate this aspect.

Whether these differences are due to differences in the immune systems of these hosts or to differences in the parasitological infections is not known. It is possible that in reservoir hosts, where trypanosomes appear not to be highly pathogenic these autoantibodies do not develop. In man and rabbits where marked pathological symptoms are usually seen it is believed that trypanosomes exert some kind of adjuvant effect breaking down self-tolerance with subsequent production of autoantibodies. Cattle appear to show an intermediate effect where a single infection with either T. brucei or T. congolense produces only a small transient rise in anti-liver autoantibody but repeated fly borne challenge and periodic
**Table.** Infections and autoantibody titres in hartebeests, hyaenas and lions captured in the Serengeti National Park, Tanzania

<table>
<thead>
<tr>
<th>Host</th>
<th>Infections</th>
<th>CTF Titres</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>Species</td>
<td>Tryp</td>
</tr>
<tr>
<td>253</td>
<td>Hartebeest</td>
<td>−</td>
</tr>
<tr>
<td>255</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>257</td>
<td>Alcelaphus</td>
<td>b</td>
</tr>
<tr>
<td>258</td>
<td>buselaphus</td>
<td>b</td>
</tr>
<tr>
<td>259</td>
<td>c</td>
<td>+</td>
</tr>
<tr>
<td>239</td>
<td>Hyaena</td>
<td>−</td>
</tr>
<tr>
<td>249</td>
<td>c</td>
<td>+</td>
</tr>
<tr>
<td>250</td>
<td>Panthera</td>
<td>−</td>
</tr>
<tr>
<td>251</td>
<td>c</td>
<td>+</td>
</tr>
<tr>
<td>252</td>
<td>Crocuta</td>
<td>c</td>
</tr>
<tr>
<td>254</td>
<td>crocuta</td>
<td>bc</td>
</tr>
<tr>
<td>257</td>
<td>b</td>
<td>+</td>
</tr>
<tr>
<td>258</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>259</td>
<td>c</td>
<td>+</td>
</tr>
<tr>
<td>245</td>
<td>b</td>
<td>−</td>
</tr>
<tr>
<td>246</td>
<td>bc</td>
<td>+</td>
</tr>
<tr>
<td>247</td>
<td>c</td>
<td>−</td>
</tr>
<tr>
<td>257</td>
<td>c</td>
<td>+</td>
</tr>
<tr>
<td>258</td>
<td>−</td>
<td>+</td>
</tr>
</tbody>
</table>

**Tryp** = Trypanosomes:  
\(b\) = *T. brucei*  
\(c\) = *T. congolense*  
\(T?\) = Unidentified *Trypanosoma*

**MF** = Microfilaria  
**Hep** = *Hepatozoon*  
**Piro** = Piroplasm  
**a-C\(^1\)** = anti-complementary titre  
**Anti-Wass** = anti-Wassermann autoantibody titre

Treatment with Diminazene aceturate (Berenil) results in much higher autoantibody levels (Mackenzie et al., in preparation).

The low levels of autoantibodies recorded here in hartebeest infections may be comparable to the single infections in cattle when the immunogenic stimulus is insufficient to completely break tolerance.
Acknowledgements

We wish to thank Professor Geigy for allowing one of us (ARM) to join his survey in the Serengeti National Park and for all his advice. Our thanks are due to the Director of the East African Trypanosomiasis Research Organisation for providing laboratory space for part of this work. We are extremely grateful to the Overseas Development Administration of the Foreign and Commonwealth Office and the Medical Research Council for financial support. This work was undertaken while one of us (ARM) held a Medical Research Council Scholarship for training in research methods.

References


