The results of such an experiment are recorded in Table 2. They show that liver and mast cells have a high capacity to adsorb cytophilic antibody and then specifically ^{131}I-HSA. Spleen, lymph node and red blood cells show distinctly lower values. The comparison of the results obtained with and without ethanol treatment reveals that unfixed antibody treated mast cells display a greater capacity to adsorb ^{131}I-HSA than cells treated with ethanol. The results demonstrate that mast cells, liver, spleen, lymph node and red blood cells of the albino rat treated *in vitro* with rabbit antiserum against human serum albumin are capable of specifically adsorbing ^{131}I-HSA. Moreover, the uptake of antibody by liver and mast cells was nearly ten times that of the other cells, which may at least partly be due to differences in cell size.

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R. Keller

Dermatologische Universitätsklinik,
Zurich.

E. Sorkin

Schweizerisches Forschungsinstitut,
Medizinische Abteilung,
Davos.

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Immunization against *Cryptococcus neoformans* by Capsular Polysaccharide

It has been reported[1] that crude *Cryptococcus neoformans* capsular polysaccharide when injected by the subcutaneous route was incapable of protecting mice against a lethal challenge dose of the parent organism. Purified polysaccharide, on the other hand, elicited a minimal protective response which was markedly increased by coupling to an ion exchange resin adjuvant. Both polysaccharides were obtained by direct recovery from cell-free culture medium. More recently it was shown indirectly that the protective antigen appeared to be concentrated in an area of the capsule immediately surrounding the cell wall[2]. It was therefore of interest to determine if fractionation of the capsule by repeated grinding would provide antigens with increased stimulatory properties.

C. neoformans, isolated *BRI*, was grown in asparagine medium at 28° for 4 days. The cells were killed (0·5 per cent formalin for 24 h), separated from the culture medium by centrifugation, and washed five times with saline solution. Afterwards, the cells were ground in a ball mill (flint balls) at 4° C for 24 h, washed free of loosely bound polysaccharide and reground. A total of five such procedures were carried out. The cells after the last grinding were approximately 90 per cent decapsulated when examined microscopically in india ink. Less than 1 per cent of the cells appeared to be ruptured. Polysaccharide was recovered from each fraction and purified by procedures described earlier[1].

The results presented in Table 1 substantiated the earlier reported indirect evidence for the existence of an immunizing antigen in close proximity to the cell wall of this organism. When the same immunization regimen was used in a second series of animals (Table 2), it was established that the specificity of the most active fraction (Cap-5) appeared to be species specific, but not strain specific.

In a third series the polysaccharide was administered as before with the exception that spleen weights and serum hæmagglutination titres were monitored at intervals. The antibody determinations were made by a modification of the technique described by Landy *et al.*[3]. The results of this experiment (Table 3) complement the protection studies by demonstrating that antibody stimulation as reflected by these parameters appeared to be responsible, at least in part, for the observed protective effect. Whether this finding was due entirely to a qualitatively different antigen or represented merely a concentration effect is beyond the scope of this communication.

Table 1. Effect of Mechanically-Fractionated *Cryptococcus neoformans BRI* Capsular Polysaccharide Administration on Survival to Homologous Challenge

Group	Polysaccharide from	Survivors on post-Challenge days 10	20	30	$P <$
1	Original CFF	13/20	0/20	0/20	
2	Cap-1	18/20	4/20	0/20	
3	Cap-2	18/20	5/20	0/20	
4	Cap-3	20/20	8/20	2/20	
5	Cap-4	20/20	9/20	4/20	0·05
6	Cap-5	20/20	16/20	11/20	0·03
7	Control	12/20	0/20	0/20	

Groups consisted of white, male (12–14 gm), *ICR* mice. Each animal received a single 600 μg dose of polysaccharide intravenously (control = 0·2 ml. saline). All mice were challenged with 10^3 cells of *C. neoformans BRI* seven days after antigen dose. Data are the arithmetic means of duplicate experiments.
Abbreviations: CFF, cell-free filtrate; Cap-1, supernatant from first grinding; Cap-2, supernatant from second grinding; etc.

Table 2. Effect of *Cryptococcus neoformans BRI* Cap-5 Polysaccharide Administration on Survival to Heterologous Challenge

Group	Challenge organism	Dose	Survivors on post-Challenge days 10	20	30	$P <$
1	*C. neoformans* TRE		20/20	18/20	13/20	0·05
2	Control	$10^{4.5}$	15/20	2/20	0/20	
3	*C. neoformans* BRY	$10^{5.2}$	20/20	18/20	14/20	0·05
4	Control		16/20	3/20	0/20	
5	*Staphylococcus aureus* KMG	Approx.	9/20	0/20	0/20	
6	Control	10^8	9/20	0/20	0/20	
7	*Klebsiella pneumoniae* LIM	Approx.	30/20	0/20	0/20	
8	Control	10^7	2/20	0/20	0/20	

Groups consisted of white, male (12–14 gm), *ICR* mice. Immunization and challenge schedule, same as Table 1. Data are the arithmetic means of duplicate experiments. All organisms are isolates from human cases.

Table 3. Primary Response to Cap-1 and Cap-5 Polysaccharides from *Cryptococcus neoformans BRI* as Reflected by Spleen Weights and Hæmagglutination Titres

Day after antigen dose	Cap-1 Mean spleen weight (per cent difference from control $\pm \sigma$)	Cap-1 Reciprocal of hæmagglutination titre (pool)	Cap-5 Mean spleen weight (per cent difference from control $\pm \sigma$)	Cap-5 Reciprocal of hæmagglutination titre (pool)
1	− 0	< 10	0	10
3	0	< 10	0	10
5	0	< 10	0	10
7	0	< 10	+ 1·7 ± 0·45	20
10	+ 0·8 ± 0·15	< 10	+ 7·8 ± 0·30	40
14	+ 1·2 ± 0·29	10	+ 12·2 ± 1·1	40

Groups consisted of twenty-five white, male (12–14 g), *ICR* mice inoculated with 600 μg of polysaccharide. At intervals animals were bled from the abdominal aorta (ether anæsthetic) for serum and killed to obtain spleens.
Abbreviations: Cap-1, first grinding; Cap-5, fifth grinding; σ, standard deviation.

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Hans H. Gadebusch

General Medical Research Section,
U.S. Veterans Administration Hospital,
Ann Arbor,
Michigan.

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ANATOMY

Olfactory Relationships of the Diencephalon

The dominant part played by olfaction in the behaviour of lower mammals has long been appreciated, but it is only comparatively recently that the underlying physiological mechanisms have begun to be elucidated. There is now a considerable body of evidence implicating cer-