allergen standardization, a problem which has always been particularly difficult in relation to house dust mite and mite extracts.

The component isolated from *D. farinae* in this study should prove valuable in skin testing, for house dust mite allergen standardization and for use in in vitro tests such as the RAST. RAST studies with mite extracts have been found to correlate well with the results of skin tests and positive clinical diagnoses of house dust mite hypersensitivity[16,17]. The above results therefore, lead us to conclude that we have isolated an important allergen from extracts of *D. farinae*. *D. farinae* and *D. pteronyssinus* show antigenic and allergenic cross-reactivity[5,17] and the *D. farinae* component isolated by precipitation with tridacnin, cross reacts with a constituent in *D. pteronyssinus* extract. Tridacnin should therefore be equally useful for allergen extraction studies with the latter species. Studies are in progress in an attempt to characterize the tridacnin-reacting mite component and to further examine its role in clinical hypersensitivity to mites.

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Effect of Dipyridamole on Human Platelet Phospholipids

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Summary. The effect of dipyridamole on platelet phospholipids has been studied. After the platelet incubation with dipyridamole, a 38% reduction in the concentration of the sphingomyelin and a 21% increase in phosphatidyl choline were produced.

Dipyridamole seems to have an antithrombotic effect as proved in vitro[1] as well as in vivo[2]. The dipyridamole action mechanism may be related to the maintainance of a high level of plasmatic adenosin[3], either by hindering the circulating adenosine deaminase action[4] or because its incorporation into the platelets is altered[5-7]. However, ROZENBERG and WALKER[8] suggest that adenosine-induced inhibition is not mediated extracellularily or intracellularily, but by a process within the platelet membrane.

On the other hand, we have observed in our laboratory that, when the platelets are incubated in vitro with dipyridamole, an alteration of their membrane electrokinetic potential is produced[9]. As the phospholipids participate in the electrokinetic potential of the platelet through their negatively charged phosphate groups[10], we have thought it interesting to evaluate the action of dipyridamole on the platelet phospholipids so as to verify if after platelet incubation with dipyridamole, any phospholipid alteration is obtained that could be related to the variations of the electrokinetic potential or that could influence the ionic transport through the platelet membranes.

Material and methods. All the methods applied have been described in an earlier paper[11] and they are, in principle, as follows: The blood sample to be used is drawn with trisodium citrate solution 3.8% (9/1). It is centrifuged at 900 rpn ($\simeq$ 100 *g*) and 4 °C for 15 min, thus obtaining a platelet-rich plasma (PRP). Subsequently, a platelet precipitate is obtained by differential centrifuga-

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Effect of dipyridamol on the platelet phospholipids

	Major phospholipids (%)					
	Platelet rich plasma (0.9 ml) plus saline (0.1 ml)		Platelet rich plasma (0.9 ml) plus dipyridamol (final concentration 4 µg/ml)			
	$\bar{x}$ ($n = 17$)	SD	$\bar{x}$ ($n = 16$)	SD	t	P
Sphyngomyelin	14.85	2.56	11.12	2.59	3.96	< 0.01
Phosphatidyl-choline	41.40	4.79	44.75	5.54	1.77	< 0.05
Phosphatidyl inositol	5.77	2.50	5.02	3.42	0.68	> 0.1
Phosphatidyl serine	10.89	3.29	10.56	3.98	0.24	> 0.1
Phosphatidyl ethanolimine	25.98	3.32	28.46	5.32	1.53	> 0.1

tion and, after washing with isotonic saline solution, they are resuspended in a volume of saline similar to that of the initial plasma.

To effect the extraction of phospholipids, an acidified organic solvent is used once the platelets have been homogenized with perchloric acid 0.6 *M* (V/V). Then, it is centrifuged at 3000 rpm for 15 min ($\simeq$ 1500 $\times g$) and the resulting precipitate is treated with a mixture of chloroform, methanol, hydrochloric acid 0.1 *N* (20/10/0.1). After being agitated, 8 ml of hydrochloric acid 0.1 *N* is added and then centrifuged. The supernate is taken away and the platelet button undergoes another extraction following the same method.

Thin layer chromatography is used to evaluate the phospholipids using silical-gel at 250 μ. Previous to their use the chromatographic plates are washed with a mixture of chloroform, methanol, acetic, water (50/30/8/4), and immediately after they are dried at 110 °C for 1 h. The same mixture used to wash the plates is used as a solvent. After the application of the sample (0.1 ml), it is left running for 45 min, then dried, and finally developed with phosphomolybdic at 20% or rhodamine 6G at 0.1% in ethanol at 96%.

To quantify fractions obtained, they are separated by scraping and their contents of phosphorus multiplied by 25 to obtain phospholipids.

Results and discussion. In our experiences 0.9 ml of a normal plasma, with its platelets adjusted to 270,000 mm^3, was incubated with a dipyridamole solution in isotonic saline solution, giving a final concentration of 4 μg/l. After 10 min incubation at room temperature, the platelets were separated by differential centrifugation and the concentrations of the total phospholipids and the major phospholipid fractions were evaluated. In the control sample the 0.1 ml of dipyridamole solution was substituted by 0.1 ml of saline solution. The assays with and without dipyridamole have been performed on aliquots of the same platelet rich plasma.

It was observed (Table) that, after the platelet incubation with dipyridamole, a 38% reduction in the concentration of the sphingomyelin and a 21% increase, which is likely to be compensatory for the reduction, in the phosphatidyl choline, were produced. Undoubtedly, an alteration of the platelet phospholipids could influence the maintainance of the ideal conditions for the normal functioning of the platelet membranes[12]. All this is more suggestive if it is taken into account that, as recently described by SCHICK[13], small hydrolysis of the platelet phospholipids can notably influence the release reaction.

It has also been possible to assert in our laboratory that aspirin, a drug with an antitrombotic action as well, reduces in vitro as well as in vivo the sphingomyelin rate[14]. The possible correlation between both actions is, for the moment, unknown to us.

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The Effects of Tryptophol on Immune Responses and its Implications Toward Trypanosome-Induced Immunosuppression

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Summary. Daily administrations of tryptophol to laboratory rodents resulted in significantly depressed antibody production to heterologous red blood cell challenge, but did not alter cellular-mediated responses to oxazalone. These results suggest that trypanosome-produced tryptophol may account for the immunodepression observed during trypanosomiasis.

The phenomenon of trypanosome-induced immunodepression has been reported to occur in experimental rodent infections[3–5], and in natural human infections[6]. The mechanisms of such immunologic abberations are ill-understood. One suggestion is that the trypanosomes elaborate a product which is directly suppressive to the host's immune apparatus[7]. Indeed, LONGSTAFFE[8] reported that live trypanosomes added to cultures of normal lymphocytes depressed mitogenic responses to phytohemagglutinin.

This paper reports upon an investigation into the immunosuppressive properties of tryptophol (indole-3-ethanol). This substance has been demonstrated to be synthesized by *Trypanosoma brucei gambiense*[9] and it has been reported that the metabolism of tryptophan is significantly increased in *T.b. gambiense* infected rats[10]. Laboratory animals, mice and field voles, were given repeated injections of tryptophol and the following immunologic parameters were investigated: 1. humoral levels and cellular production of antibodies to heterologous erythrocytes; 2. cell-mediated responses to oxazalone; 3. spleen cell thymidine-uptake; and 4. the course of Ehrlich's ascites tumor growth.

Materials and methods. The laboratory animals used in this investigation consisted of the field vole, *Microtus montanus*, and white mice. *M. montanus* were obtained from our colony maintained at Tulane University in New Orleans, Louisiana. CD-1 mice were obtained from Charles River Laboratories, Wilmington, Massachusetts. Daily

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