among the brachiopods of the order Strophomenida in the sense of Williams (*12, 13*). On the other hand, *Tentaculites* does not look like a brachiopod, as it is not a bivalved organism in the usual sense of the word. If there were indisputable evidence of a hinged operculum (*3*) it might be plausible to suggest it as homologous to one of the brachiopod valves. But contrary to the findings of Blind (*3*), with none of the specimens of *Tentaculites* that I studied is there any evidence of an operculum—attached or unattached.

The suggestion that *Tentaculites* might be the spines of a brachiopod was broached in 1831 by von Buch (*14*), who incorrectly considered them to be the spines of *Leptaena lata*. The strophomenide shell fabric has been reported from broken productid spines (*12*), and Lyashenko (*1*) noted the similarity of the tentaculitid wall structure to that of the brachiopod spine. But the major problem with the spine hypothesis is the fact that tentaculitids are not found associated with any brachiopods from which they were clearly a part. Conversely, no brachiopods have been found with spines that are grossly similar to *Tentaculites*. The absence of undoubted muscle scars [the drawings of Lardeux (*15*) notwithstanding] limits a clear interpretation of the shell as a housing for an entire organism, although some phoronids are able to move freely within their unmineralized tubes (*16*). The tubes of free-living annelid worms are open at both ends.

Perhaps the *Tentaculites* were specialized brachiopods or perhaps they were calcified phoronid tubes. Neither of these suggestions is compelling, but neither are the alternatives in the literature. Interestingly, the brachiopods and the phoronids have often been placed together (along with the bryozoans) in the phylum Tentaculata, the validity of which is supported by recent biochemical data (*17*). On the basis of shell fine structure and mineralogy, *Tentaculites* are clearly more closely related to articulate brachiopods than to mollusks or annelids. Sadly, there are as yet no known living phoronids having calcified tubes with which to compare them.

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Lymphocyte Ecto-5′-Nucleotidase Deficiency in Agammaglobulinemia

Abstract. *Fresh peripheral blood lymphocytes from eight patients with congenital agammaglobulinemia demonstrate reduced ecto-5′-nucleotidase activity when compared to the mean activity of normal subjects and patients with other forms of immunoglobulin deficiency. A specific defect of ecto-5′-nucleotidase is further suggested by normal values for lymphocyte ecto-adenosinetriphosphatase and ecto-nonspecific phosphatase. The data provide evidence for an enzyme deficiency in this X-linked, B lymphocyte deficiency syndrome.*

Inherited enzyme deficiencies in purine nucleotide degradation have been associated with immune deficiency states involving disorders of thymus-dependent (T) and thymus-independent (B) lymphocyte function. The absence of adenosine deaminase (E.C. 3.5.4.6) has been associated with a severe immunodeficiency syndrome involving defective B and T lymphocyte activity (*1*), whereas the absence of the next enzyme of the degradative pathway, purine nucleoside phosphorylase (E.C. 2.4.2.1), is characterized by a T lymphocyte deficiency (*2*). A third possible disorder, the decreased activity of 5′-nucleotidase (E.C. 3.1.3.5) on the external surface of the lymphocyte, has been observed in a heterogeneous group of patients with hypogammaglobulinemia (*3, 4*).

To distinguish whether 5′-nucleotidase deficiency is associated with a specific form of lymphocyte dysfunction, we measured lymphocyte ecto-5′-nucleotidase in patients with hypogammaglobulinemia subdivided into definable disease categories. All eight males with congenital agammaglobulinemia have deficient lymphocyte ecto-5′-nucleotidase with values ranging from 30 to 48 percent of normal activity (Fig. 1). One other male infant, who did not meet all the criteria for congenital agammaglobulinemia, had low ecto-5′-nucleotidase ac-

Table 1. Activity of plasma membrane bound enzymes in lymphocytes. Intact lymphocytes from normal subjects or patients with congenital agammaglobulinemia were prepared as described in Fig. 1. Ecto-adenosinetriphosphatase was assayed in 0.25×10^6 cells with 150 m*M* NaCl, 0.75 m*M* $MgCl_2$, 8 m*M* KCl, 1 m*M* 5′-adenosine triphosphate, and 80 m*M* tris-HCl, *p*H 8.5, in a total volume of 150 μl at 37°C for 60 minutes. After the incubation period, the reaction mixture was cooled to 4°C and centrifuged for 3 minutes. One hundred microliters of supernatant was removed for an inorganic phosphate assay (*14*). The reaction was linear with time to 120 minutes and with cell number. Ecto-nonspecific phosphatase activity was measured in 0.5×10^6 lymphocytes in a reaction mixture which contained 150 m*M* NaCl, 70 m*M* tris-HCl at *p*H 7.0, and 4 m*M* *p*-nitrophenylphosphate in a total volume of 275 μl at 37°C for 30 minutes. The reaction was stopped with 700 μl of 0.4*M* NaOH. Change in optical density at 410 nm measured the enzymatic release of *p*-nitrophenol. The assay was linear with time to 30 minutes and with cell number from 0.1×10^6 to 1.0×10^6 lymphocytes.

Subjects	Ecto-enzyme					
	Adenosinetriphosphatase [μmole hour^{-1} (10^6 cell)$^{-1}$]			Nonspecific phosphatase [nmole hour^{-1} (10^6 cell)$^{-1}$]		
	Value	Range	*N*	Value	Range	*N*
Normal	0.12	0.03 to 0.26	10	36.1	12.5 to 70.2	9
Congenital agammaglobulinemia	0.12	0.06 to 0.22	5	40.1	10.5 to 103.0	9

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Table 2. Activity of 5′-nucleotidase in intact and lysed lymphocytes. Fresh, intact, peripheral blood lymphocytes were separated and assayed for ecto-5′-nucleotidase as described in Fig. 1. The lymphocyte suspension was centrifuged at 1200*g* for 10 minutes. The lymphocyte pellet was frozen at −70°C for 1 to 4 weeks. The lymphocytes were lysed by freeze-thawing and the supernatant dialyzed in NaCl (150 m*M*) and 100 m*M* tris-HCl, *p*H 7.5, for 8 hours at 4°C. The dialyzed supernatant was then assayed for 5′-nucleotidase. The difference in ecto-5′-nucleotidase activity in lymphocytes from normal subjects and patients with congenital agammaglobulinemia is statistically significant ($P < .02$) in both intact and lysed lymphocytes (unpaired two-tailed Student *t*-test).

Subjects	Lymphocyte 5′-nucleotidase [nmole hour^{-1} (10^6 cell)$^{-1}$]	
	Intact	Lysate
	Normal	
A	16.4	6.4
B	12.1	6.3
C	18.3	6.5
D	13.3	6.7
	Congenital agammaglobulinemia	
A	8.5	5.9
B	7.6	5.3
C	7.5	5.5
D	5.4	3.3
E	5.7	3.9
F	4.4	5.6

tivity. In contrast, lymphocytes assayed from all patients with selective immunoglobulin A (IgA) deficiency and six of seven patients with common variable hypogammaglobulinemia have lymphocyte ecto-5′-nucleotidase values similar to lymphocytes from normal subjects (Fig. 1). Ecto-5′-nucleotidase activity is essentially the same in (erythrocyte) E-rosetting and non-E-rosetting lymphocyte subpopulations in both normal and agammaglobulinemic patients (*5*).

The enzyme 5′-nucleotidase catalyzes the hydrolysis of purine 5′-nucleotides [the monophosphates of adenosine (AMP), inosine, guanosine, and xanthosine] to their respective nucleosides (adenosine, inosine, guanosine, and xanthosine) by the reaction: nucleoside monophosphate + $H_2O \rightarrow$ nucleoside + P_i. This enzyme is present in many tissues and can be detected in different subcellular fractions including the plasma membrane where it functions as an ecto-enzyme with enzymatic activity facing the external medium (*6*). Previous studies of 5′-nucleotidase in intact lymphocytes have established it as an ecto-enzyme which regulates the uptake of AMP into lymphocytes by converting the nontransportable nucleotide to its readily transported nucleoside, adenosine (*7*).

Other plasma membrane marker proteins were measured to determine if the activity of ecto-5′-nucleotidase is a selective deficiency of a single plasma membrane enzyme. Ecto-adenosinetriphosphatase (E.C. 3.6.1.3) and nonspecific phosphatase (E.C. 3.1.3.2) values in lymphocytes from congenital agammaglobulinemia patients were similar to the values observed in normal subjects or patients with other forms of hypogammaglobulinemia (Table 1). These data support a selective decrease of 5′-nucleotidase activity.

To distinguish whether the deficiency occurs in the plasma membrane or whether it involves other forms of 5′-nucleotidase, we compared lysates from the 5′-nucleotidase–deficient lymphocytes and normal lymphocytes. The marked difference in plasma membrane enzyme activity is not as apparent in cell lysates (Table 2). These data suggest that the deficiency is restricted to the plasma membrane form of the enzyme.

Additional studies of the lymphocyte ecto-5′-nucleotidase with decreased activity were designed to determine if this enzyme is structurally altered (Table 3). The *p*H optimum of the deficient ecto-enzyme is indistinguishable from the normal *p*H optimum of 7.5. The ecto-5′-nucleotidases with normal and decreased activity are inhibited by 70 and 82 percent of the control value, respectively, by adenosine 5′-α,β-methylene diphosphonate, a specific inhibitor of 5′-nucleotidase (*8*). Both enzymes demonstrate hyperbolic kinetics during initial velocity studies and have essentially the same value of the Michaelis constants for AMP.

Table 3. Properties of lymphocyte ecto-5′-nucleotidase. Intact lymphocytes were prepared and assayed for ecto-5′-nucleotidase as described in Fig. 1. For the measurement of *p*H optimum, a range of values from *p*H 6.5 to 9.5 was used. The Michaelis constant (K_m) for AMP was measured by using initial velocity studies with AMP concentrations ranging from 5 to 50 μM. Double reciprocal plots were used to determine the Michaelis constants of ecto-5′-nucleotidase in lymphocytes from two normal subjects and two patients with congenital agammaglobulinemia. The effect of adenosine 5′-α,β-methylene diphosphonate (AOPCP) (*8*) on ecto-5′-nucleotidase activity was evaluated in lymphocytes from both types of subjects.

Subjects	*p*H optimum	K_m for AMP (μM)	Inhibition by 25*M* AOPCP (%)
Normal	7.5	27, 26	70
Congenital agammaglobulinemia	7.5	29, 20	82, 85

The discovery of lymphocyte ecto-5′-nucleotidase deficiency in patients with agammaglobulinemia is important for several reasons. First, a subgroup of patients with agammaglobulinemia has the enzyme deficiency associated with a dysfunction of B lymphocytes and an X-linked inheritance (*9*). This may represent a third inborn error of purine metabolism associated with an immuno-

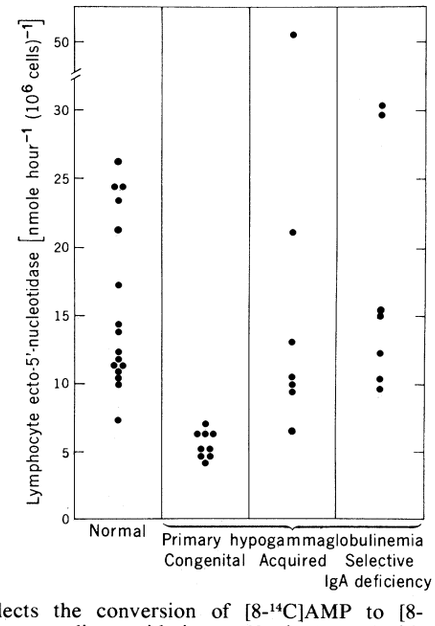

Fig. 1. Peripheral blood lymphocytes were obtained by separation from fresh heparinized blood on a Ficoll-Hypaque gradient (*14*). Cells were washed three times and suspended in Hanks balanced salt solution without calcium or magnesium. The enzyme assay was carried out immediately after lymphocyte preparation. Incubations were performed in duplicate at 37°C in a reaction volume of 100 μl containing approximately 0.25×10^6 lymphocytes. The lymphocyte suspension was added to a mixture containing 150 m*M* tris-HCl, *p*H 8.5, 2.5 m*M* $MgCl_2$, and 200 μM [8-^{14}C]AMP. The mixture was incubated for 30 minutes and the reaction was stopped by heating at 85°C for 4 minutes. Twenty-five microliters of the reaction mixture was spotted with nonisotopic adenosine, inosine, and AMP, each 1 mg/ml, on Whatman 3MM chromatographic paper. The AMP, adenosine, and inosine were separated by high-voltage electrophoresis in 50 m*M* sodium borate, *p*H 8.9, for 30 minutes at 250 mA and 400 V. Ultraviolet spots corresponding to inosine and adenosine were cut out and the radioactivity was measured in a liquid scintillation spectrometer system. Enzyme activity measured in this manner reflects the conversion of [8-^{14}C]AMP to [8-^{14}C]adenosine and [8-^{14}C]inosine. The enzyme assay was linear with time to 60 minutes and with lymphocyte numbers ranging from 0.05×10^6 to 0.5×10^6. The mean value for the normal lymphocytes is 15.0 nmole per hour per 10^6 lymphocytes, while the mean value for congenital agammaglobulinemia is 5.7 nmole per hour per 10^6 lymphocytes.

deficiency syndrome, which is genetically distinct from the first two diseases described (*10*). Further studies will be necessary to confirm the distribution of the enzyme deficiency within the spectrum of humoral immunodeficiency diseases. Second, the enzyme deficiency and lymphocyte dysfunction may be etiologically related, as appears to be the case for the first two diseases (*11*). Such an etiologic relation between the enzyme deficiency and the immune dysfunction is supported by the observation that a subgroup of patients with chronic lymphocytic leukemia have a deficiency of lymphocyte ecto-5′-nucleotidase (*5, 12*). Patients with this disease may also develop a deficiency of serum immunoglobulins and recurrent infections similar to those seen in primary hypogammaglobulinemia (*13*). Finally, lymphocytes lacking 5′-nucleotidase are unable to take up AMP as a result of an inability to hydrolyze this compound to adenosine (*7*). It is possible that this decreased ability to degrade 5′-nucleotides to their catabolic products may impair B lymphocyte function and explain the occurrence of some manifestation of agammaglobulinemia. Therefore, the discovery of another disorder of the immune system associated with a specific deficiency of purine nucleotide degradation provides additional clues for the role of purine catabolism in the regulation of immune function.

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Isopod and Insect Root Borers May Benefit Florida Mangroves

Abstract. *Far from threatening the persistence and geographic extent of red mangrove (*Rhizophora mangle*) in Florida, wood-boring marine isopods may aid the plant to survive wave action by initiating branching of aerial prop roots. Evidence for a recent, sudden increase in density or range of one such isopod,* Sphaeroma terebrans, *is anecdotal and weak. Insect damage to mangrove aerial roots even before they descend to the water is at least as great as that wrought by isopods and also causes root branching. Aerial and submarine damage combine to stimulate root initiation so that, for every root produced aerially by the tree, at least 1.4 roots reach the substrate. Similar responses to herbivory, which have been reported for other plants, suggest that herbivores may both benefit and harm plants, and that their impact may be more difficult to assess in specific instances than has been realized.*

Herbivory is traditionally viewed as detrimental to plants (*1*), with victims suffering at least loss of biomass and possibly death or deformation through destruction of a growing tip or unique stem. Red mangrove (*Rhizophora mangle*) swamps of Florida's west coast, with their prominent prop roots originating from normal aerial branches often high in the trees, are said to be in grave danger from a wood-boring marine isopod, *Sphaeroma terebrans*, which attacks the roots (*2, 3*) once they descend to the water. "An ecocatastrophe of serious magnitude" is heralded: "The shoreline of the mainland and of the mangrove islands is gradually shrinking . . . *Sphaeroma* has already eliminated much of the protective outer edge of this great mangrove stand. It threatens to eliminate much more . . ." (*2*). "The isopod does not kill the tree, but sometimes, without the support of its prop roots, a red mangrove topples into the water during storms" (*3*).

A sudden, spontaneous environmental disaster of this magnitude would indeed be both a curious and a serious matter,

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