of the points he has raised have been dealt with in previous published exchanges in the *Journal of Investigative Dermatology*.

1. Dr. Hearing suggests that our inability to demonstrate hydroxylation of tyrosine by mammalian "tyrosinase" is related to the use of a concentration of tyrosine sufficient to produce total substrate inhibition. We are not aware of documentation of *total* substrate inhibition in any enzyme system. In a previous study [1] we used a range of tyrosine concentrations and found that this had no effect on the inability of mammalian "tyrosinase" (dopa oxidase) to hydroxylate tyrosine. In addition, we repeated experiments in our current study, using a similar range of tyrosine concentration, without effect on our data. Conversely, hydroxylation of tyrosine by *mushroom* tyrosinase was easily demonstrable [2] using tyrosine concentrations equal to those employed with our mammalian "tyrosinase" experiments.

2. Dr. Hearing's alleged inability to obtain adequate "tyrosinase" concentration using our method is of no significance and has no bearing on our results. His assay system was ambiguous, and his method of differentiating peroxidase from "tyrosinase" activity was without value for reasons cited previously [3]. In our previous experiments [2] and in the current study, our enzyme preparation (mammalian "tryosinase") had good specific activity towards dopa, using nonambiguous assay methods.

3. Rx value of an enzyme will vary with many factors, including its polymeric form, binding to other proteins [4], and polymerization time used for the gel; pore size will vary with polymerization time. Therefore, Rx value cannot reliably be used as a parameter of enzyme identification. It is not clear why the dopa-positive bands he found at the Rx values cited in our current study did not represent "tyrosinase." We do not understand why Dr. Hearing could suggest that our dopa-positive bands represented hemoglobin or peroxidase, since we clearly stated that these bands were diaminobenzidine-negative, excluding these possibilities.

4. Dr. Hearing should be aware that electrophoretic characteristics of melanoma peroxidase cannot be determined, since this peroxidase is firmly membrane-bound. We make this point in our rebuttal to his previous paper [3].

The only way that the controversy about "tyrosinase" can be resolved is by step-by-step crossverification of results, with exchange of starting materials and intermediates between laboratories with conflicting data, as well as the use of nonambiguous methods of enzyme assay. In recent correspondence we have offered to participate in this crossverification with Dr. Hearing. It is most unfortunate that he has declined this offer.

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AUTOSENSITIZATION TO DNA

To the Editor:

We read with great interest the article by Pinnas et al [1] reporting decreased T-cells and positive skin immunofluorescence in a patient with sensitization to DNA and autologous whole blood. We have previously reported similar findings in 2 patients with clinical and laboratory features consistent with autoerythrocyte sensitization [2]. Both patients had decreased T-cell counts. Immunofluorescence of involved and uninvolved skin revealed irregular granular deposits of IgM at the dermal-epidermal border of involved skin only in one patient. Both patients had slightly depressed complement levels during periods of clinical activity. We look forward to additional reports on skin immunofluorescence and laboratory immunologic parameters in other patients with similar purpuric disorders.

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REPLY

To the Editor:

Drs. Krain, Levin, and Schultz describe 2 patients with decreased serum complement levels during the period of ecchymoses, although only 1 of their patients had immunofluorescent deposits of IgM without complement at the basement membrane in the ecchymotic area. Furthermore, this patient had evidence also of an associated immunologic disorder, angioimmunoblastic lymphadenopathy in which T-cell deficiency has been reported (Frizzera G, et al, Am J Med 59:803, 1975). Deposits of IgG and IgA have been reported along the basement membrane in a patient with autoerythrocyte sensitization and lymphoid interstitial pneumonia (Decoteau W, et al, Arch Intern Med 134:519, 1974).

An early case of autoerythrocyte sensitization showed that histamine could precipitate purpura and might be important in the pathogenesis (Schulman N, et al, Clin Res 7:216, 1959). More recently, histamine has been shown to facilitate immune complex deposition in patients with vasculitis (Braverman I, Yen A, J Invest Dermatol 65:105, 1975).

In autoerythrocyte sensitization, lesions could be induced by intracutaneous injection of synthetic bradykinin but not by histamine (Leiba H, Isr J Med Sci 8:67, 1972). Our findings of IgM and C_3 at the dermaepidermal junction in a patient with autosensitization to DNA was associated with other immunologic abnormalities but not with an obvious underlying immunologic disorder. More recently, IgM and C_3 have been found in vessel walls in the upper dermis in a patient with purpura associated with erythema chronicum migrans (Gross G, et al, Arch Dermatol 115:873, 1979). We have also found immunoglobulin and complement deposits within alveolar hyaline membranes in idiopathic respiratory distress syndrome and group B streptococcal sepsis during the early neonatal period (Pinnas J, et al, Pediatrics 63:557, 1979).

It is apparent that a number of mechanisms associated with inflammation may share a final common pathway associated with immune complex deposition in the involved organ.

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