ing, such as already demonstrated in turtles, may take place (*8*). In any event, the present data show that innate perceptual differences can be useful in the study of closely related as well as more distantly related forms, and that the analysis of the chemical perceptual mechanisms involved should consider evolution and ecology.

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References and Notes

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2. G. M. Burghardt, *Psychon. Sci.* **4**, 37 (1966).
3. More complete details concerning the subjects, the experimental procedures, and the results are recorded in G. M. Burghardt, thesis, University of Chicago (1966).
4. To check the reliability of the testing procedure, 20 trials (10 water and 10 nightcrawler) were later run on different snakes with a second observer independently timing attack latencies and counting tongue flicks. This second observer did not know which extract was being presented. The average tongue-flick count discrepancy for a given trial was less than 1 and the average latency discrepancy less than 0.5 second. The rank correlation of the 20 trials was highly significant (r_s=.997).
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9. Assisted by NIH grants MH 776 and MH 13375. I thank E. Lace and other naturalists at the Palos Division, Cook County Forest Preserve, P. Allen, and H. Campbell for providing the gravid females and newborn young; E. H. Hess, E. Klinghammer, G. S. Reynolds, T. Uzzell, and D. Wake for assistance.

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Effect of Neuraminidase on Isozymes of Alkaline Phosphatase and Leucine Aminopeptidase

Law's report (*1*) on the effect of neuraminidase on isozymes of alkaline phosphatase and leucine aminopeptidase would be of greater interest if sufficient details had been given to allow the reader to evaluate the validity of the data presented. The effects of neuraminidase on the fast-moving alkaline phosphatase isozyme of human serum are well known from the work of Robinson and Pierce (*2*), a paper not cited by Law. These authors found that the degree of retardation of the fast-moving phosphatase isozyme is dependent upon neuraminidase concentration. With high concentrations of neuraminidase, the migration of the fast component was retarded to a point much closer to the origin than that of the normal slow-moving isozyme.

Law reports the effects of only one concentration of neuraminidase and contends that the presence of neuraminic acid accounts for the difference between the isozymes. This conclusion is dubious unless it can be shown that higher concentrations of neuraminidase or treatment for a longer time do not further alter the mobility of the fast-moving isozyme.

Although Law mentions in a footnote the activity at *p*H 5.1 of the neuraminidase preparation he used, he fails to mention the *p*H he used in his experiments on the serums. [Neuraminidase preparations from Sigma Chemical Co. are either from *Vibrio cholera* or *Clostridium perfringens* (*3*); the enzymes from both sources are most active in a *p*H range of approximately 4.5 to 6 (*4*).] Assuming that Law treated the serums at a *p*H appropriate for effective neuraminidase activity, one would like to know the effect of this *p*H on the mobility of the enzymes not treated with neuraminidase. In view of the fact that Law mentions no control of normal serum treated with the buffer used for neuraminidase treatment, how may one determine whether the alteration of mobility is due to the action of neuraminidase or to the effect of lowered *p*H on the enzymes? One might also wonder whether there is any protease activity in the neuraminidase preparation.

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The report of Robinson and Pierce, noted by Etzler, points out that genetically determined protein variants can differ by carbohydrate components—neuraminic acid, for example. My report of studies of chickens supports their suggestion. Under the conditions of my experiments with neuraminidase, two genetically controlled isozymes of both alkaline phosphatase and leucine aminopeptidase were affected differently.

The fast-moving forms were retarded in mobility to a point similar to the slow forms which were unaffected by the neuraminidase treatment. Addition of 0.5 mg of neuraminidase directly to 1 ml of plasma did not appreciably alter the *p*H of the plasma; thus all the change in migration of the fast forms of the enzymes was ascribed to the neuraminidase treatment. The amount of neuraminidase present was sufficient to provide conditions for complete reaction with all the enzymes in 1 ml of plasma even if the *p*H was not within the approximate range of 4.5 to 6, as mentioned by Etzler. Other electrophoresis experiments with buffers covering a range of *p*H from 4.0 to 9.5 have shown that the fast form is always anodal to the slow form. With appropriate low *p*H buffers in the gels, the slow forms migrate toward the cathode, and the fast forms migrate toward the anode. This further shows the net charge differences between the two molecular forms of both enzymes.

A thorough study of the number of neuraminic acid residues on each of the enzyme molecules and comparison with the similar enzymes in man would be rewarding. One contrast between chickens and man is that no secretion of blood group substance has been reported in chickens. The relationship of alkaline phosphatase in man and the secretion of blood group substance is well known, and it has been cited by Robinson and Pierce.

The main points of my report were that genetically determined variants of both enzymes were directly related in all samples tested and that the neuraminidase treatments have led to the suggestion that genetic control of a common carbohydrate component of the enzymes is responsible for the relationship.

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