| First enzyme* | Second enzyme* | Nanomoles of sialic acid released from two glycoproteins | | | |
|------------------|-------------------|---|--------|--------------------------------|--------|
| | | Total from both enzymes | | Contribution of second enzymet | |
| | | Human | Bovine | Human | Bovine |
| NWS | Water | 38‡ | 9 | 0 | 0 |
| | NWS | 46 | 14 | 8 | 5 |
| | ws | 50 | 41 | 12 | 32 |
| ws | Water | 50 | 46 | 0 | 0 |
| | NWS | 56 | 41 | 6 | -5 |
| | ws | 56 | 50 | 6 | 4 |
| | | 1 | 1 | 1 | 1 |

 TABLE 1. Residual substrate after treatment with NWS

* An amount of 0.05 ml of bovine α -GP (20 mg/ml) or human α -GP (4 mg/ml) in 0.5 M phosphate-buffered saline; 0.05 ml of enzyme in both cases. Pretreatment, 4 hr; second enzyme, 2 hr.

† Calculated from the reaction with water as a base line of zero for activity of the second enzyme.

‡ Average of three samples for the group with NWS as first enzyme.

enzymes with the bovine α -GP is provided by the data in Table 1. Assuming that the neurotropic virus enzymes release only acetylneuraminic acid, all the glycolylneuraminic acid should be

intact when the reaction has reached completion. Therefore, a considerable amount of substrate should be available to the WS enzyme after exhaustive reaction with the NWS enzyme. This is what the data suggest, with a 3.5:1 ratio of total product released by WS as compared with NWS (50:14). If WS releases all available sialic acid, and NWS releases only acetylneuraminic acid, the theoretical ratio of substrate concentrations would be 100% to 36%, or ca. 3:1. The quantity of sialic acid liberated from the human α -GP was about the same for both the WS and NWS enzymes. In addition, it is shown that nearly all of the enzyme-susceptible sialic acid of both substrates is released by the WS enzyme when it is used as the first enzyme.

Qualitative demonstration of the enzyme products is currently in progress. However, the data presented here suggest that the enzymes of the neurotropic variants of type A influenza virus WS are not active upon glycolyl-substituted neuraminic acid. The possible influence of different linkage partners for the sialic acids in these substrates has not been considered. It is not possible at this stage to speculate what relationship such a substrate specificity might have to the neurotropic properties of these strains.

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ERRATUM

Nucleotide Accumulation Induced in Staphylococcus aureus by Glycine

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Volume 89, no. 4, page 1125, Table 1: change the heading of the sixth column from "Glucose" to "Glutamic acid."