**Abstract**

Helicobacter pylori has been reported to agglutinate erythrocytes and to bind to various other cells in a sialic acid-dependent way. The binding was inhibited by sialyllactose or fetuin and other sialylated glycoproteins. The specificity apparently requires bacterial growth on agar, since we found that it was lost after growth in the nutrient mixture Ham's F12. Instead, the bacteria bound with high affinity and in a sialic acid-dependent way to polyglycosylceramides of human erythrocytes, a still incompletely characterized group of complex glycolipids. Bacteria grown in F12 medium were metabolically labelled with 35S-methionine and analysed for binding to glycolipids on thin-layer chromatograms and to glycoproteins on blots after electrophoresis, with human erythrocyte glycoconjugates in focus. There was no binding to simpler gangliosides including GM3 or sialylparagloboside, or to a mixture of brain gangliosides. In contrast, polyglycosylceramides of human erythrocyte membranes bound at a pmol level. The activity was eliminated by mild acid treatment, mild periodate oxidation or sialidase hydrolysis. Erythrocyte proteins as well as a range of reference glycoproteins did not bind except band 3, which was weakly active. However, this activity was resistant to periodate oxidation. These results indicate a second and novel sialic acid-recognizing specificity which is expressed independently of the previously described specificity.