

and Nussenzweig, V. (1991) *Cell.*, **65**, 1117-1125. (3) Previato, J. O., Andrade, A. F. B., Vermelho, A., Firmino, J. C. and Mendonça-Previato, L. (1990) *Mem. Inst. Oswaldo Cruz, Suppl.*, I **85**, 38.

S10.11

Gal α 1-4Gal-Specific Cell Binding Activity in *Streptococcus suis*

S. Haataja^{a,b}, K. Tikkanen^c and J. Finne^{a,c}

^aDepartment of Medical Biochemistry; ^bDepartment of Chemistry, Institute of Biomedicine, University of Turku, SF-20520, Turku, Finland and ^cDepartment of Biochemistry and Biotechnology, University of Kuopio, SF-70211 Kuopio, Finland.

Streptococcus suis causes meningitis and septicemia in pigs and occasionally in humans. Novel adhesion activities including galactose-dependent and sialic acid-dependent binding were discovered by screening *S. suis* strains by hemagglutination. The hemagglutination of the galactose recognizing strain 628 was inhibited by galactose and *N*-acetylgalactosamine at millimolar concentrations. Inhibition experiments with di- and oligosaccharides revealed that the structures containing the Gal α 1-4Gal β 1-sequence were inhibitors at micromolar concentrations (Haataja, S., Tikkanen, K., Liukkonen, J., François-Gerard, C. and Finne, J. (1993) *J. Biol. Chem.*, **268**, in press). TLC-overlay assays showed binding to GbO₃, but not GbO₄ or GbO₅, in contrast to *E. coli* containing the P adhesin that showed preferential binding to the latter two glycolipids. The preference for GbO₃ was also indicated by the high binding activity of the *S. suis* 628 strain with erythrocytes containing high amount of this glycolipid (P⁺ erythrocytes, rabbit erythrocytes) whereas erythrocytes lacking P antigens (p erythrocytes) were not agglutinated. The adhesin also binds the P₁ structure, as revealed by binding to P₁-active glycoproteins and neoglycoproteins.

The detailed binding specificity was determined with deoxy- and deoxy-fluoro-derivatives of galactose and was found to differ from that of *E. coli*. Thus, the *S. suis* adhesin resembles the *E. coli* P adhesin in its specificity towards blood group P-related structures but shows a distinct specificity towards the P⁺ and P₁ structures. Like with *E. coli*, a variant Gal α 1-4Gal adhesin differing in its binding specificity was recently also identified.

S10.12

Is the Salicylic Acid Glucoside Bioactive

J. Hennig¹, J. Malamy², D. F. Klessig², G. Gryniewicz³, J. Indulski³

¹Institute Biochemistry and Biophysics, Polish Academy of Sciences, Warszawa, Poland; ²Waksman Institute, Rutgers, The State University of New Jersey, Piscataway, USA; ³Pharmaceutical Research Institute, Warszawa, Poland.

Many plants are able to resist pathogen attack by localizing the pathogen to a small region near the infection site, where a necrotic lesion later forms. This type of resistance is termed the hypersensitive response (HR). Following the HR many plants show a heightened ability to resist a secondary attack by the same or even unrelated pathogens. This acquired resistance is seen not only near the infection site, but

throughout the plant. The systemic appearance of acquired resistance (SAR) is accompanied by many molecular and physiological changes, including the induction of genes encoding several families of proteins called the pathogenesis-related (PR) proteins.

We showed that SA is in fact a natural product of tobacco, synthesized by the resistant cultivar Xanthi nc but not by the near isogenic susceptible cultivar Xanthi following infection by tobacco mosaic virus (TMV). Enhanced levels of SA were detected in both inoculated and uninoculated leaves, correlating with the systemic appearance of PR proteins and acquired resistance. Based on these and related findings, SA has been proposed to act as a signal in the induction of PR genes and in the establishment of SAR. Recently, we demonstrated that levels of a sugar conjugate of SA, salicylic acid β -glucoside (SAG), increased in parallel with SA levels after TMV infection. SA applied to tobacco leaves was also found to be rapidly converted to this conjugated form. Thus both SA and SAG appear to be synthesized *de novo* during the resistance response, with SA being readily converted to SAG.

Since SA is rapidly converted to salicylic acid β -glucoside (SAG) in tobacco, we have attempted to assess the role of SAG in pathogenesis by application of chemically synthesized SAG to tobacco leaves. SAG was as active as SA in induction of PR-I gene expression. This induction was preceded by a transient release of SA, which appears to occur in the extracellular spaces. The existence of a mechanism that releases SA from SAG suggests a possible role for SAG in disease resistance.

S10.13

Binding Specificity of Bacterial Adhesins from *Neisseria subflava* Strains and other Bacteria to Glycolipids Incorporated into Liposomes: Comparison with TLC Binding Patterns

M. Abul-Milh, J. Ångström, H. Dezfoulian and K.-A. Karlsson

Department of Medical Biochemistry, University of Göteborg, S-413 90 Göteborg, Sweden.

Different strains of *Neisseria subflava* were tested for their binding specificities on artificial surfaces (TLC plates) using mixtures of glycosphingolipids from various sources as well as purified glycolipids and compared to the corresponding binding patterns obtained with a liposome assay into which purified glycolipids had been incorporated. Glycolipids of the ganglio series were found to be efficient receptors in both assay systems although with varying binding strengths which may be ascribed to differences in epitope presentation. An earlier TLC investigation (1) revealed different affinities of *Neisseria subflava* to LacCer containing hydroxy- or non-hydroxyceramide. However, in the liposome assay this difference appears insignificant.

We will further present an investigation of enteropathogenic *E. coli* (EPEC) wild type strain binding to glycosphingolipids using the above mentioned assays, and compare their binding specificities with those of uropathogenic wild type and cloned *E. coli* strains (2).

1. Nyberg, G., Stromberg, N., Jonsson, A., Karlsson, K.-A. and Normark, S. (1990) *J. Infect. Immun.*, **58**, 2555 – 2563. 2. Ångström, J., Abul Milh, M., Lanne, B. and Karlsson, K.-A. (1993) submitted.