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Munc18a promotes vesicle tethering in neuronal and non-neuronal fusion

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Sec1-Munc18 (SM) proteins cooperate with SNAREs {SNAP [soluble NSF (N-ethylmaleimide-sensitive factor) attachment protein] receptors} to promote membrane fusion in eukaryotic cells. Studies of Munc18-1 in neurotransmission suggest that SM proteins accelerate fusion kinetics primarily by activating zippered trans-SNARE complexes. However, Munc18-1 has also been found important in the exocytosis of non-neuronal cells. Here we investigate the function of Munc18a in reconstituted fusion reactions mediated by distinct sets of exocytic SNAREs. We show that Munc18-1 has a new role in promoting proteoliposome tethering. In the three different fusion reactions examined, Munc18a-dependent tethering requires the N-peptide in syntaxins but displays divergence regarding the requirement for SNAP-25-like molecules. In addition, tethering is preserved under inhibitory conditions that abolish both trans-SNARE complex formation and lipid mixing, indicating that Munc18-1 catalyzes tethering in a step preceding trans-SNARE zippering and activation.

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Comparative glycomics in *Caenorhabditis elegans*

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Caenorhabditis elegans is a simple model organism composed of 959 developmentally well-defined somatic cells. This nematode is a good model for tissue development, microbial infection and innate immunity. It is infected by over 40 microbial pathogens most of which are also human pathogens. We have studied a series of glycosylation deficient *C. elegans* strains that are resistant to a series of bacterial infections. The bus-4 strains are resistant to bacterial infection caused by *Microbacterium nematophilum*, *Yersinia pestis*, *Yersinia pseudotuberculosis* and *Staphylococcus aureus*. Here, we describe our glycomics study of the bus-4 reference strain in comparison to its parent strain, N2 Bristol. Using mass spectrometry based strategies as well as expression array analyses, we investigated the N- and O-glycomes of the bus-4 strain and the impact that altered glycosylation processes have on expression and ultimately bacterial infection. We have found that the N-glycome is essentially unperturbed. However, the *C. elegans* mucin oligosaccharides are significantly altered especially in the abundance of charged species and overall glycomer distributions. Also, two key mucins are greatly upregulated. Expression analysis reveals an altered early innate immune factor pattern in the uninfected state resembling that of wild-type nematodes in the state of infection by gram positive bacteria. Changes in elements of secretory system control were found. Overall, our study reveals likely mechanisms that may be involved in resistance to *S. aureus* infection.

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