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Glycosylation regulates CD38 assembly on the cell surface

Miki Hara-Yokoyama
Tokyo Medical and Dental University, Japan

Many proteins have their functions on the cell membranes or organelle membranes. To understand the function on the membranes, it is important to elucidate the cell-surface assembly. The leukocyte cell-surface antigen CD38 is a type II transmembrane glycoprotein and has four N-glycosylation sites. CD38 is the major NAD^+ glycohydrolase in mammals and its ecto-enzyme activity is involved in calcium mobilization. CD38 also acts as a lipid raft-dependent signalling molecule to promote cell proliferation or death. CD38 forms a tetramer on the cell surface but the structural basis and the functional significance of tetramerization have remained unexplored. We identified the interfaces contributing to the homophilic interaction of mouse CD38 by site-specific crosslinking on the cell surface with an expanded genetic code based on a crystallographic analysis. A combination of the three interfaces enables CD38 to tetramerize: One interface involving the juxtamembrane α-helix is responsible for the formation of the core dimer which is further dimerized via the other two interfaces. This dimerization of dimers underlies the catalytic activity and the localization of CD38 in lipid rafts. The N-linked glycosylation sites are found to be located in strategic positions to prevent further self-association of the tetramer. Accordingly, the glycosylation is likely to ensure the function of CD38 by regulating the cell-surface assembly.

Biography

Miki Hara-Yokoyama has received her PhD in 1986 at the Department of Biophysics and Biochemistry, Faculty of Science, Tokyo University. She worked at the Department of Physiology in Nihon University School of Dentistry at Matsudo as a Research Associate (1986-1995) and as a Lecturer (1995-2001). Then, she moved to the Department of Biochemistry in Tokyo Medical and Dental University (TMDU) and worked as a Lecturer (2001-2004). Currently, she is an Associate Professor of the department. Her research interests include regulation of protein assembly on the membrane by glycosylation or lipid-environment.

m.yokoyama.bch@tmd.ac.jp

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