Dr. Toby Jenkins　講演会

Stability, Permeability and Toxin Interaction with Surface Tethered Lipid Vesicles

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日時：平成 19 年 5 月 15 日　　16 時 10 分-17 時 40 分
場所：長崎大学総合教育研究棟 207 講義室

バース大学（英国）の Jenkins 先生が長崎を訪問される機会に高分子学会九州支部主催外国人講演会を開催します。カプセル化された水溶性 BODIPY®蛍光色素を取り込んだ大きなユニラメラ DMPC ベシクルの表面固定化と Surface Plasmon Resonance (SPR)および Surface Plasmon field-enhanced Fluorescence (SPFS)による評価に関する最近のご研究についてご講演いただきます。多数のご出席下さいますようご案内申し上げます。

Stability, Permeability and Toxin Interaction with Surface Tethered Lipid Vesicles

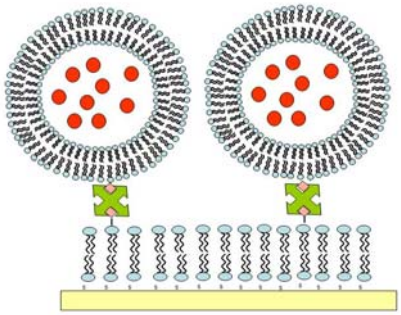
Presented by Dr. Toby Jenkins

(Department of Chemistry, University of Bath)

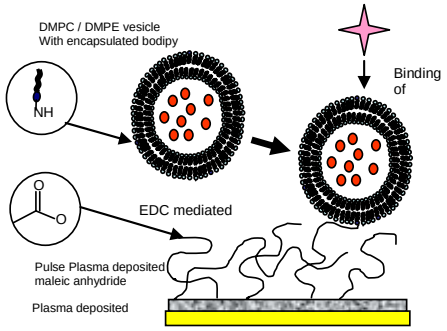
Date: 15th May, 2007 (16:10-17:40)

Place: Room 207, General Education and Research Building, Nagasaki University

This paper describes recent results from an investigation into the surface immobilization of large unilamellar DMPC vesicles containing an encapsulated, water soluble bodipy fluorophore. Two attachment methods are described: biotin-streptavidin Self-assembled Monolayer and the creation of soft plasma film 'cushions', with vesicle being attached via EDC coupling. The surface immobilized vesicles were studied using combined Surface Plasmon Resonance (SPR) and Surface Plasmon field-enhanced Fluorescence (SPFS). The two attachment methodologies are sketched below.



SAM / Lipid bilayer vesicle support architecture



Plasma polymer hydro gel vesicle support system

The stability of the vesicle was studied by quantifying the leakage rate of fluorophore from within the vesicles in real-time and fitting to a model based on the partial partition of fluorophore into the bilayer and the diffusion controlled diffusion from the surface. The effect of adding a number of membrane disrupting toxins, including heamolytic snake venom, phospholipase A_2 and cholera toxin was studied, with both binding of protein and the lysis event being studied simultaneously, but independently by SPR and SPFS.

The methodology developed in this work allows for measurement of toxin-membrane interactions in a fluid membrane environment. This has given information on both the mechanism of toxin interaction and on new methods for blocking binding events. We show that Eu III salts block the interaction of the cholera αβ toxin with GM1 receptor units on the membrane, leading to the possibility of developing new therapeutic methods for treating certain gut based infections such as cholera.