Z503 **The Role of Glycoconjugates in Fas-FasL Interaction and Fas Mediated Apoptosis**

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The interaction of Fas(CD95) and its ligand (FasL) triggers apoptosis and is involved in the regulation of immune responses. Fas mediates apoptosis by FasL trimer binding on Fas and death domain of Fas oligomerizations. Our aims are to prove the role and importance of glycosylation in Fas-FasL interaction and Fas mediated apoptosis. Fas has two N-linked and one O-linked glycoconjugates. We show that mutation of N-glycosylation and O-glycosylation site has effect on protein secretion and protein expression level. Deglycosylated mutants (NM2, NM12, OM) were not secreted to media and reduced expression level. When both N-glycan were deleted, Fas lost its ligand binding activity. We demonstrate that sialic acid treatment inhibits Fas-FasL binding and Fas mediated apoptotic cell death. We think that this is due to the competition between FasL and sugar. We observed that the effect of sialic acid on Fas-FasL interaction is linkage specific. And we show that sialic acid exists in glycoconjugates of Fas and is required for Fas-FasL binding. When sialidase was treated to Fas-Fc, ligand binding activity of Fas was reduced.

Z504 **Apoptosis in MCF-7 Cells Treated with PKC Inhibitors and Daunorubicin**

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Apoptosis usually was induced by PKC inhibitors or daunorubicin in MCF-7 cell line. The staurosporine of PKC inhibitor was induced apoptosis in the cytoplasm but 1-(5-isoquinolinylsulfonyl)-2-methylpiperazine(H-7) effected to more wide areas, such as cytoplasm and nucleus. Daunorubicin induced apoptosis inboth cytoplasm and nucleus, but most cells were observed cell death after daunorubicin first, then, PKC inhibitors treating to the cells. In the results of using by southern blot, apoptosis were occurred a small number of cells and DNA was separated more high molecules treating with PKC inhibitors or daunorubicin for 2 1/2 hrs than those of treating it for 24 hrs. But DNA was separated low molecules in a large number of cells treating with PKC inhibitors and daunorubicin for 24 hrs. Therefore, it suggests that DNA in the apoptosis was more split in the long time exposure than short time treating PKC inhibitors or daunorubicin.