49 cases stuided with genotypes and serotype, genotype 1b, 1b/2b, 2a, 2a/2c and 2b were 51, 2.0, 34.6, 8.1 and 4.0%. There were no significant difference in responce to alpha-interferone tretment of HCV infection with the subtype 1b or 2a. The serotypes ?type and ?type were 57.1% and 42.8%, respectively and mached with genotypes in 85.7%, and seemed to be easy to perform. This study demonstrates that immunoblot assay is more useful to screen the HCV infection and RT-PCR-Hybridization test is choice of confirming the HCV infection in patients with positive immunoblot results, and that serotype test was preferred to genotype for monitoring progression or response to treament.

G106 NKT CELL ASSOCIATED IMMUNE RESPONSE: ENHANCED TUMOR VACCINE EFFECT

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NKT cells are a subset of novel population, which is believed to perform immune-regulatory functions. We found that activation of NKT cells at the time of immunization can greatly enhance cytotoxic killer cell associated immune responses. In this report, tumor vaccine against B16 melanoma was directly investigated under the condition where NKT cells were specifically stimulated. Treatment of alpha-galactsylceramide (*α*GalCer), a specific NKT cell antigen, at the time of tumor vaccination showed great enhancement of viability upon live tumor (B16) injection in mouse model.

G107 Prostaglandin E2 induce hypermethylation of the 5'regulatory region of IFN- gene by elevation of the intracellular cAMP and NO in the human Jurkat T-cells

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Expression of cytokine by T lymphocytes is a highly balanced process, involving stimulatory and inhibitory intracellular signalling pathways. Prostaglandin E_2 (PGE_2) induced a strong inhibition of IFN- mRNA expression and this inhibition was assumed to be related to intracellular cAMP concentration and nitric oxide generation. However, it remains to be clarified how these factors inhibit production of IFN-. In this context, we examined the relationship of the inhibitory effects of PGE_2 and DNA methylation on INF- gene expression in the human Jurkat T-cells. The CpG islands within the TATA proximal regulatory element of the IFN- gene were methylated by treatment of the Jurkart T-cells with PGE_2. The methylation was not induced by treatment of the Jurkart T-cells with db-cAMP or SNAP alone. However, IFN- gene was methylated by treatment of the Jurkart T-cells in combination with db-cAMP and SNAP. These results suggested that PGE_2 inhibit IFN- production by inducing hypermethylation of IFN- gene through elevation of the intracellular cAMP and NO in the human Jurkat T-cells.

G108 Role of SRG3 in the Inhibition of Glucocorticoid-Induced Apoptosis of Immature Thymoma Cells in Response to TCR/CD3 Signaling

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Some evidence has shown that TCR activation of Ras/MEK/ERK pathway plays a critical role in the inhibition of glucocorticoid (GC)-induced apoptosis. We have previously demonstrated the significant correlation between the expression level of SRG3 and the GC sensitivity of developing thymocytes. In this study, we examined the effect of TCR/CD3 signaling on the expression of the SRG3 gene using murine immature thymoma cell lines. TCR/CD3 signaling resulted in a dramatic decrease in SRG3 expression. Specifically, TCR/CD3 downregulation of the SRG3 gene was mediated by Ras/MEK/ERK and PI3K, but not Ral.GDS pathway. We also found that binding of E47/HEB complex to the E-box element in the SRG3 minimal promoter was inhibited by Id3 inhibitor of E proteins. Finally, introduction of mutations into the E-box element in the SRG3 promoter completely abrogated the TCR/Ras responsiveness of the SRG3 promoter.

G109 Notch1 confers a Resistance to Glucocorticoid Developing Thymocytes by Down-regulating SRG3 Expression

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We have previously reported that SRG3 is required for the glucocorticoid-induced apoptosis in the S49.1 thymoma cell line. Activation of Notch1 was shown to induce glucocorticoid resistance in thymocytes. However, the specific downstream target of Notch1 conferring thymocytes glucocorticoid resistance is currently unknown. We found that the expression level of SRG3 was critical in determining glucocorticoid sensitivity in developing thymocytes. The expression of SRG3 was also downregulated by the activated form of Notch1 (NotchIC). The promoter activity of the SRG3 gene was also downregulated by NotchIC. Expression of transgenic SRG3 resulted in the restoration of glucocorticoid sensitivity in thymocytes expressing transgenic Notch1. These results suggest that SRG3 is the downstream target of Notch1 in regulating glucocorticoid sensitivity of thymocytes.

G110 Peripheral T Cells Become Sensitive to Glucocorticoids- and Stress-induced Apoptosis in Transgenic Mice Overexpressing SRG3

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Immature double positive thymocytes are sensitive to glucocorticoid-induced apoptosis, while mature single positive T cells are relatively resistant. Thymocytes seem to acquire resistance to glucocorticoids during differentiation into mature single positive thymocytes. However, detailed knowledge concerning what determines the sensitivity of thymocytes to glucocorticoids and how glucocorticoid-sensitivity is regulated in thymocytes during development is lacking. We have previously reported that the murine SRG3 gene (for SWI3 related gene) is required for the glucocorticoid-induced apoptosis in a thymoma cell line. Herein, we provide results suggesting that the expression level of SRG3 protein determines the glucocorticoid-sensitivity of T cells in mice. SRG3 associates with the GR in the thymus but rarely in the periphery. Transgenic overexpression of the SRG3 protein in peripheral T cells induces the formation of the complex and renders the cells to become sensitive to glucocorticoid-induced apoptosis. Our results also show that blocking the formation of the SRG3-glucocorticoid receptor complex with a dominant negative mutant form of SRG3 decreases glucocorticoid-sensitivity in thymoma cells. In addition, mice