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The role of miR-155 in the apoptosis of human lymphoma cell induced by CIK cells

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Objective: To observe the effect of cytokine-induced killer cells (CIK) on the apoptosis of human lymphoma cells (Raji and BJAB), and explore the role of micro RNA-155(miR-155) in this process.

Methods: MiR-155 was determined by real time quantitative PCR and the apoptosis was detected by flow cytometry in Raji and BJAB cells. Psi CHECK2-CEBPB 3'-UTR containing the binding site of miR-155 was constructed, and then transfected into Raji and BJAB cells. Luciferase activity of CEBPB (CCAAT/enhancer binding protein beta) was determined with the assistance of dual luciferase report system.

Results: CIK cells could promote Raji and BJAB cells apoptosis (t=3.634, P<0.05; t=3.976, P<0.05), and increase the expression of miR-155 in Raji by (6.87±0.19) fold (t=2.787, P<0.05), meanwhile, in BJAB cells by (5.06±0.25) fold (t=3.513, P<0.05). Moreover, miR-155 inhibitor might block this process (t=3.842, P<0.05; t=4.016, P<0.05). Furthermore, miR-155 mimics induced Raji and BJAB cells apoptosis (t=4.239, P<0.05; t=3.477, P<0.05). MiR-155 targeted at the site of CEBPB 3'-UTR, and CIK cells could decrease the luciferase activity of Raji cells by (42.89±2.06)% (t=3.281, P<0.05); meanwhile, in BJAB cells by (37.02±1.98)% (t=4.933, P<0.05).

Conclusion: CIK cells could enhance human lymphoma Raji and BJAB cells apoptosis by upregulating miR-155, which may provide a new database to elucidate lymphoma cell therapy using CIK cells.

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Critical role of all-trans retinoic acid in stabilizing human nature regulatory T cells under inflammatory conditions

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Recent studies have demonstrated that thymus-derived naturally-occurring CD4+Foxp3+ regulatory T cells (nTregs) in both human and mouse are unstable and dysfunctional in the presence of pro-inflammatory cytokines. All-trans Retinoic Acid (atRA), the active derivative of vitamin A, has been shown to regulate Treg and T effector cell differentiation. It is hypothesized atRA stabilizes human nTregs under inflammatory conditions. atRA prevents human nTregs from converting to Th1 and/or Th17 cells and sustains their Foxp3 expression and suppressive function in vitro or in vivo following encountering with IL-1 and IL-6. Interestingly, adoptive transfer of human nTregs pre-treated with atRA significantly enhanced their suppressive effects on xeno-graft versus host diseases (xGVHD) and atRA- but not rapamycin-pretreated nTregs sustained the functional activity against xGVHD after stimulating with IL-1/IL-6. atRA suppresses IL-1R upregulation, accelerates IL-6R downregulation, and diminishes their signaling events as well as prevents the upregulation of Stub1 on Foxp3+ cells following IL-1/IL-6 stimulation. atRA also increases histone acetylation on Foxp3 gene promoter and CpG demethylation in the region of Foxp3 locus (TSDR). These results strongly implicate that nTregs primed with atRA may represent a novel treatment strategy to control established chronic immune-mediated autoimmune and inflammatory diseases.

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