Injury by Human Complement Causes Large Membrane Lesions that Reseal in IL-4-treated Porcine Endothelial Cells

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CONCLUSIONS

- Based on NR results, significant LDH loss from EC would not be expected in IL-4-protected cells. However, similar amounts of LDH are lost from cells treated with human complement (in HS) regardless of whether they were pretreated with IL-4 or not.
- Thus, IL-4 does not prevent the formation of lesions in EC during incubation with complement, but does help protect the cells from killing by complement.
- IL-4-protected EC appear to have initially leaky membranes, but are able to reseal the membrane lesions caused by complement and avoid cell death.

MATERIALS AND METHODS

- Treatment of EC with IL-4 and Complement: During the IL-4 pretreatment, EC were incubated with either medium alone (1% FBS/DMEM) or with medium containing 5 mg/ml IL-4 for 48 hrs. The EC were then incubated with solutions containing either 20% human serum (HS) as a source of antibody and complement or 20% heat-inactivated human serum (HI-HS) for 2 hrs. Complement solutions were then replaced with medium (1% FBS/DMEM) for a recovery period of 0, 2, 4, or 22 hrs.

RESULTS

IL-4 induces protection of EC from complement killing, but LDH still leaks out of EC

- IL-4 does not prevent PI uptake during complement treatment

REFERENCES

2. Luke Larkman et al., “IL-4 protects pannus blood vessel endothelial cells and does not alter expression of syndecan-1 or the endothelin-1 receptor, but does increase expression of CD59,” Arthritis and Rheumatism 44 (January 2001): 730-736.
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