NON-LYMPHOID CULTURED CELLS POSSESS A SYSTEM CONTROLLING CELLULAR COMPATIBILITY

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We show that various non-lymphoid cultured cells can activate the production of both soluble and membrane-bound cytotoxic factors in response to direct contact with cells of a different kind. Accumulation of cytotoxic factors in the medium was detected already 1 h after contact of K562 and L929 cells or after contact of L929 cells with purified membranes of K562 cells. No cytotoxic effect was observed when K562 and L929 cells were cultivated in two sections of a plastic flask separated by a membrane with 0.4 μm pores, i.e. when the direct cellular contacts were excluded but the exchange of soluble factors was not restricted.

Analysis of proteins recovered from the culture medium collected after cocultivation of K562 and L929 cells has revealed the presence of three cytotoxic factors including TNF-α. TNF-α and/or immunologically related proteins, but not Fas-ligand or lymphotoxin, were also accumulated in membranes of K562 and L929 cells shortly after these cells had been allowed to contact each other. The cytotoxic factors expressed by non-lymphoid cells trigger apoptosis of target cells.

Isolation of clones of L-929 cells capable of expressing TNF-α has been reported previously [1-3]. However, these selected clones produced TNF-α either constantly or under permanent selection for TNF-α resistance. Hence, there was no question of fast activation of TNF-α (or other cytotoxic factor) expression in response to external stimuli such as contact with other cells. In contrast, in our case fast activation of TNF-α expression by different non-lymphoid cells in response to contact with cells of a different type was observed. This, in turn, signifies that non-lymphoid cells possess receptors capable of discriminating cells of a different kind. Furthermore, non-lymphoid cells must also possess molecular mechanisms connecting these receptors with systems regulating expression of TNF-α and possibly of other presently uncharacterized cytotoxic proteins. In other words, our results suggest that individual non-lymphoid cells possess a molecular mechanism regulating cellular compatibility.
REFERENCES

