Activated T lymphocytes are polarized, motile cells. The leading edge of a motile T cell is highly sensitive to chemokines [1, 2]. Chemokines can induce lymphocyte polarization with redistribution of the adhesion molecules, ICAM-1, ICAM-3, CD44, and CD43 to the cell uropod, a membrane protrusion at the end of a migrating lymphocyte opposite the direction of locomotion [3-5]. These events have been shown to play an important role in cell-cell interactions and in migration of immune cells toward sites of inflammation [6].

Serrador et al. [7] demonstrated that membrane-cytoskeleton interactions facilitate redistribution of these adhesion receptors to the uropod in lymphocytes treated with chemokines. Cells treated with RANTES or MCP-1 or with an ICAM-3 antibody exhibit redistribution of ICAM-3 and the cytoskeletal protein, moesin, to the tips of uropods. The interaction of moesin with ICAM-3 in T lymphocytes is mediated through the intracellular region of ICAM-3, and correlates with the degree of cell polarity. Myosin II is also localized in the neck of the uropod, and a myosin-disrupting drug that prevents uropod formation inhibits moesin redistribution to the uropod tip and subsequent moesin-ICAM-3 association.

The data of Serrador et al. suggest that during chemokine-induced lymphocyte polarization, the cytoskeletal protein moesin is important for the redistribution of adhesion molecules to the cellular uropod (Fig. 1). This model raises interesting questions concerning what signaling pathways are involved in connecting chemokine receptor(s) with the cellular cytoskeleton.
REFERENCES


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