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Opinion Article

Cell-Extracellular Matrix Interactions: The Biomechanics of Cellular Adhesion and Migration

Luca Moretti*

Department of Biomedical Devices, University of Bologna, Bologna, Italy

DESCRIPTION

Cellular behavior, including adhesion and migration, is intricately influenced by mechanical forces generated within the cell and its environment. The Extra Cellular Matrix (ECM), a dynamic network of proteins and polysaccharides that surrounds and supports cells, plays a critical role in these processes. The interaction between cells and the ECM governs various cellular functions, from tissue development and wound healing to cancer metastasis. This article explores the biomechanics of cellular adhesion and migration, shedding light on how cells physically interact with the ECM and how mechanical forces drive these important processes. The ECM is a complex, multi-component structure that provides both structural support and biochemical signals to cells. Composed of proteins such as collagen, fibronectin and laminin, as well as glycolsaminoglycans, the ECM serves as a scaffold that influences the mechanical properties of tissues. Cells adhere to the ECM through specialized receptors called integrins, which anchor the cells to ECM components and transmit mechanical and biochemical signals across the cell membrane. These interactions not only help cells maintain their shape but also regulate cellular behaviors such as migration, proliferation and differentiation. Cellular adhesion to the ECM is a highly regulated process that involves focal adhesions-multi molecular complexes that connect integrins on the cell surface to the actin cytoskeleton inside the cell. When a cell adheres to the ECM, integrins cluster together and form these focal adhesions. Within the focal adhesions, integrin-linked kinase and other signaling molecules transduce mechanical signals from the ECM into biochemical responses that can alter gene expression and cellular behavior. From a biomechanical perspective, tension within the focal adhesions is important for their formation and maintenance. This force facilitates the continuous remodeling of the ECM, allowing the cell to maintain or adjust its attachment. This tension-driven process is not passive; it actively shapes the ECM and influences tissue structure and function. The biomechanical aspects of migration are governed by the forces generated by the cytoskeleton. Actin filaments polymerize at the leading edge of the cell, pushing the membrane forward, while myosin motors contract actin filaments at the rear to generate traction. The balance between these forces at the front and rear of the cell determines the direction and efficiency of migration. Meanwhile, the ECM influences cell migration by providing cues such as substrate stiffness, topography and ligand density. For instance, cells tend to migrate more efficiently on stiffer substrates, a phenomenon known as durotaxis. The interaction between cells and the ECM is reciprocal, not only do cells respond to mechanical forces exerted by the ECM, but they also apply forces back onto the ECM, leading to its remodeling. This dynamic interaction is critical in tissue development and maintenance, where the mechanical properties of the ECM change in response to cellular activity. During wound healing, for example, fibroblasts contract the ECM to close the wound, applying mechanical forces that modify the tissue structure. Similarly, in cancer, tumor cells remodel the ECM to facilitate their migration and invasion into surrounding tissues. Matrix Metallo Proteinases (MMPs), enzymes secreted by cells, degrade ECM components, enabling cells to migrate through tissues and spread to other parts of the body.

CONCLUSION

Cellular adhesion and migration are fundamental to many physiological processes and their regulation is heavily influenced by biomechanical interactions with the extracellular matrix. The ECM not only serves as a scaffold for cells but also provides the mechanical and biochemical signals that guide cellular behavior. Over viewing the biomechanics of these interactions can offer insights into tissue development, repair and disease progression, particularly in cancer and fibrosis. Continued study into the molecular and mechanical mechanisms governing cell-ECM interactions potentials to uncover novel therapeutic targets for diseases that involve aberrant cellular adhesion and migration, such as cancer metastasis and wound healing disorders.

Correspondence to: Luca Moretti, Department of Biomedical Devices, University of Bologna, Bologna, Italy, E-mail: morettil@gmail.com

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