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日本機械学会北海道支部 バイオメカニクス懇話会
第43回講演会

(共催：日本機械学会北海道支部)

主査 大橋 俊朗

下記の要領にて第43回講演会を日本機械学会北海道支部特別講演会との共催として開催いたします。皆様のご参加をお待ちしております。

記

日時：2023年8月17日(木)，11:00～11:45

場所：北海道大学工学部工学部 A1-17

講演：

「Microenvironmental and mechanical factors on epigenetic regulation of cell homeostasis and regeneration」

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Abstract:

Microenvironmental and mechanical factors may influence cellular functions and signal transduction through epigenetic modifications. Different blood flow patterns on shear stress can affect cell cycle and inflammation in arterial endothelial cells (aECs) through histone deacetylase (HDAC) and trimethylation on lysine 9 of histone 3 (H3K9me3). Sudden transition of flow patterns from veins to arteries cause "peel-off" of venous ECs (vECs) and leads to vessel restenosis. Under arterial laminar shear stress (ALS), the vECs become round with decreased H3K9me3 and increased VCAM-1 expressions. Inhibition of HDAC activity in aECs caused similar ALS-induced inflammation and cell loss as observed in vECs, whereas the HDAC activator prevents ALS-induced peel-off and reduces VCAM-1 in vECs. Epigenetic modifications also regulate Schwann cells (SCs) during peripheral nerve injury and repair. Phenylbutyrate inhibits HDAC3 expression and activity in inflamed SCs, reducing pro-inflammatory cytokine secretion, and promoting axonal regrowth and remyelination in regenerating nerve. Furthermore, we utilized a nanoscale chitosan-deposited surface to induce spheroid formation of adipose-derived stem cells and revealed HDAC5 nucleus translocation, changes in HAT activities, and trimethylation of H3K4 and H3K9 via single-cell-RNA sequencing. Therefore, mechanical and morphological changes can drive the epigenetic control of damaged cells, as well as to reprogram the rescue cells.

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