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Homogeneous 2D and Alignment of Cardomyocytes in Dilated Cardiomyopathy Revealed by Intravital Heart Imaging

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In contrast to hypertrophic cardiomyopathy, there has been reported no specific pattern of cardiomyocyte array in dilated cardiomyopathy (DCM). This might be because it has been assessed only on heart tissue sections two-dimensionally (2D). To address this issue, we sought to evaluate cardiomyocyte alignment in DCM three-dimensionally (3D) using intravital heart imaging techniques. We observed cardiomyocytes of membrane fluorescent reporter mice up to 100μm depth by an intravital imaging system with two-photon microscopy. On each 2D image taken at 1μm interval, the angles of cardiomyocytes from a vertical line were measured and their distribution was plotted. Then the plots of all layers were merged so that layer-to-layer change of angle distribution can be visualized. In these merged plots, cardiomyocytes exhibited several peaks with a certain spread around each peak, suggesting that cardiomyocytes change their alignments by every layer in 3D and position twistedly even in a single layer. We next assessed cardiac mutant Troponin T knock-in mice as a DCM model. The angle distribution in these mice was less various within a single layer and between layers as well. These results indicates that cardiomyocytes of DCM model mice align homogeneously both in two- and three-dimensionally (Figure). To determine how homogeneous alignment contributes to cardiomyocyte contractility, we captured the motion of cultured cardiomyocytes and found that cardiomyocytes seeded on the top of linearly aligned fibres show greater motion than those seeded on randomly aligned fibres. Using intravital imaging, we have provided a first evidence of cardiomyocyte array in 3D and demonstrated that cardiomyocytes of DCM model mice align homogeneously both in 2D and in 3D. Homogeneous alignment of those mice might be the consequences of impaired cardiac function as a way to increase left ventricular contractility.

Biography

Tomoaki Higo has his expertise in molecular cardiology. He has started his career as a basic researcher by focusing on the molecular mechanisms by which DNA damage in cardiomyocytes and non-cardiomyocyte develops heart failure. He moved to Institute of Cancer Research, London in 2018 and has been investigating the relationship between cancer cell morphology and immunotherapy response at a systems level. Based on those experiences, he is now trying to understand how cardiomyocytes sense their surrounding environment and change their shape and intracellular signalling by utilising cutting-edge imaging technologies and systems biology.

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