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BMAL2 in Endothelial Circadian Rhythm and Remodeling

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ABSTRACT

Cardiovascular disease remains the number one cause of mortality in humans. An important influence in the progression of artery disease is the long-term effect of disruptions in daily patterns or circadian rhythms. For example, bad sleep and a blood pressure reading that does not fall at night worsen or even cause cardiovascular disease. The molecular mechanism that controls these activity-rest cycles is called the circadian clock and includes a key component transcription factor Bmal1. Previously, I have found that Bmal1 has an important vascular-specific role with controlling hypermuscularization and scarring in the blood vessel in a process called pathological remodeling or stiffening, using mouse models of genetic disruption. I also found that the endothelial cell layer of arteries contributed to the disease in Bmal1 knockout (KO) mice. While Bmal1 is found throughout the body, its functional and much less understood parlor, Bmal2, is more selectively expressed in the endothelium. To understand the role of Bmal2 in vascular disease, I have implemented a widely used experimental animal model of arterial ligation to induce vascular remodeling. I have ligated the left common carotid artery (LC) in two groups of mice, control wild-type mice (no genetic mutation) and the experimental Bmal2-KO (global knockout) mice. After two weeks, I isolated the LC and fixed the arteries in O.C.T. and conducted histological processing (cut cross sections with a cryotome and staining with hematoxylin and eosin). I then quantified the changes in structure in the artery using the imageJ program on digitized microscope images. My preliminary results suggest pathological thickening of the artery in Bmal2-KO mice compared to wild-type mice, thus indicating Bmal2 may have a role in vascular remodeling. Future studies will assess the endothelial specific knockout mouse of Bmal2.

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