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Thoracic aneurysm and dissection (TAAD) is a condition that puts the thoracic aortic wall under unnecessary stress. A number of new reports have suggested that arterial tortuosity may be a telltale key in predicting TAAD progression. Tortuosity, which is defined as the ratio of the actual length of the artery to the geometric length, has been shown to be proportional to aneurysm diameter. Although previous clinical studies have been conducted with a limited pool of participants and varying tortuosity indices, we plan on increasing the number of participants as well as compare various methods of tortuosity measurement to find the best indicator of TAAD progression.

Mouse models with arterial tortuosity and TAAD have been used successfully to better our understanding of aneurysm growth and intervention. Therefore, we will again use them to follow longitudinal changes in tortuosity. This project specifically focuses on three syndromes that are associated with TAAD and arterial tortuosity—Marfan Syndrome (MFS), autosomal recessive cutis laxa type 1B (ARCL1B), and Loeys-Dietz Syndrome type 1 (LDS1). We propose to determine if arterial tortuosity is predictive of aneurysmal disease outcomes in mouse models of MFS, ARCL1B, and LDS1. We will then compare our new method of monitoring TAAD progression with traditional methods. We also aim to investigate the role of TGF- , AT1r signaling and MMP activity in arterial tortuosity and aneurysm pathogenesis using mouse models of MFS, ARCL1B, and LDS1 and compare the predictive value of different measures of arterial tortuosity and length for disease outcomes in TAAD through retrospective analyses of clinical data. This involves examining MRI images of over 200 clinical patients who have MFS or LDS whose disease outcomes are known, in order to quantify the predicative value of arterial length and tortuosity.