MEF2 proteins function as regulators of gene transcription in cardiac and skeletal muscle cells. Under some circumstances the MEF2 proteins need to be inhibited to allow skeletal muscle cells to
proliferate. Also, during heart failure, MEF2 proteins are incorrectly activated leading to the pathological enlargement of the cardiac muscle cells (cardiomyocytes). In the current study we identify FoxP1
as a repressor of MEF2 activity that may function in this capacity to keep MEF2 proteins repressed when their activity is not required.