Myopalladin's Role in Cardiac Muscle Function and Disease

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Decades of research have provided fundamental insight into the human heart's structure and function. Yet, most cardiac malformations remain a mystery as scientists and clinicians continue to examine how inherited mutations and aging affect the normal biological functions of proteins associated with cardiac dysfunction. Recently, mutations in the muscle protein myopalladin have been linked to cardiomyopathy. Myopalladin and palladin belong to a family of closely related proteins that have essential, but uncharacterized roles in organizing the actin cytoskeleton. Our recent work has shown that palladin binds directly to actin and increases both the rate of actin polymerization and the stability of actin filaments. A number of mutations in myopalladin are located within the analogous actin-binding region, which suggests that a disruption in actin regulation may occur in cardiomyopathy. Thus, we hypothesized that myopalldin also binds directly to actin and increases both the rate of actin polymerization and the stability of actin filaments.