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A pathway for cardiomyopathy

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Cytoskeletal mutations have been implicated in the genetically based forms of dilated cardiomyopathy, but the mechanism by which heart failure develops is not known. In the May Nature Medicine, Mohammad Pashmforoush and colleagues from the University of California at San Diego, La Jolla, California suggest a novel developmental pathway for right ventricular dilated cardiomyopathy involving instability of the a-actinin complex.

Pashmforoush *et al*. used a murine model of cardiomyopathy and investigated mutations in the genes encoding cytoskeleton proteins. They found that disruption of the gene encoding Alp is associated with right ventricular dilation and dysfunction. *In vitro* assays showed Alp directly enhances the capacity of a-actinin to cross-link actin filaments, suggesting that loss of Alp function contributes to destabilization of actin anchorage sites in cardiac muscle.

These results implicate a-actinin-associated proteins in the onset of heart disease and provide a possible pathway that links gene defects with cardiomyopathy.

References

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