

P058 Opening up a can of worms: genetic analysis of alpha-catenin function

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Adherens junctions are important determinants of cell-cell contacts, and regulate tissue organisation and development. Alpha-catenin has a pivotal role in the assembly and maintenance of adherens junctions, but its mechanism of action is still unclear. Moreover, recent evidence has cast doubt on the long held view of its function.

We have taken a genetic approach to investigating this problem, studying alpha-catenin function in a physiologically relevant system, the morphogenesis of the *C. elegans* embryo. Our laboratory isolated a weak hypomorphic mutation affecting the actin-binding domain of the *C. elegans* alpha-catenin. This mutation, which affects a strictly conserved serine residue, reduces, but does not abolish alpha-catenin function. We have carried out a mutagenic screen to identify mutations suppress the effect of this mutation. These suppressors affect two distinct regions within the alpha-catenin actin-binding domain. Of the suppressor mutations isolated, a number of these produce premature stop codons, resulting in the truncation of the conserved C-terminal tail. Interestingly, a transgene that expresses this truncated alpha-catenin is still capable of complementing an alpha-catenin null mutation. This is in contrast to previous *in vitro* studies, which indicate that the C-terminus is essential for actin binding. Thus, while the *in vitro* data has given an insight into alpha-catenin function, our findings suggest it might not be the whole story.