

Publication

A minigene of neural agrin encoding the laminin-binding and acetylcholine receptor-aggregating domains is sufficient to induce postsynaptic differentiation in muscle fibres

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The extracellular matrix molecule agrin is both necessary and sufficient for inducing the formation of postsynaptic specializations at the neuromuscular junction (NMJ). At the mature NMJ, agrin is stably incorporated in synaptic basal lamina. The postsynapse-inducing activity of chick agrin, as assayed by its capability of causing aggregation of acetylcholine receptors (AChRs) on cultured muscle cells, maps to a 21 kDa, C-terminal domain. Binding of chick agrin to muscle basal lamina is mediated by the laminins and maps to a 25 kDa, N-terminal fragment of agrin. Here we show that an expression construct encoding a 'mini'-agrin, in which the laminin-binding fragment was fused to the AChR-clustering domain, is sufficient to induce postsynaptic differentiation in vivo when injected into non-synaptic sites of rat soleus muscle. As shown for ectopic postsynaptic differentiation induced by full-length neural agrin, myonuclei underneath the ectopic sites expressed the gene for the AChR epsilon-subunit. Altogether, our data show that a 'mini'-agrin construct encoding only a small fraction of the entire agrin protein is sufficient to induce postsynapse-like structures that are reminiscent of those induced by full-length neural agrin or innervation by motor neurons.

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