AChR phosphorylation and aggregation induced by an agrin fragment that lacks the binding domain for alpha-dystroglycan

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Agrin induces both phosphorylation and aggregation of nicotinic acetylcholine receptors (AChRs) when added to myotubes in culture, apparently by binding to a specific receptor on the myotube surface. One such agrin receptor is alpha-dystroglycan, although binding to alpha-dystroglycan appears not to mediate AChR aggregation. To determine whether agrin-induced AChR phosphorylation is mediated by alpha-dystroglycan or by a different agrin receptor, fragments of recombinant agrin that differ in affinity for alpha-dystroglycan were examined for their ability to induce AChR phosphorylation and aggregation in mouse C2 myotubes. The carboxy-terminal 95 kDa agrin fragment agrin-c95(A0B0), which binds to alpha-dystroglycan with high affinity, failed to induce AChR phosphorylation and aggregation. In contrast, agrin-c95(A4B8) which binds less strongly to alpha-dystroglycan, induced both phosphorylation and aggregation, as did a small 21 kDa fragment of agrin, agrin-c21(B8), that completely lacks the binding domain for alpha-dystroglycan. We conclude that agrin-induced AChR phosphorylation and aggregation are triggered by an agrin receptor that is distinct from alpha-dystroglycan.
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