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Involvement of Transglutaminase in Myofibril Assembly of Chick Embryonic Myoblasts in Culture

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Involvement of transglutaminase in myofibrillogenesis of chick embryonic myoblasts has been investigated *in vitro*. Both the activity and protein levels of transglutaminase initially decreased to a minimal level at the time of burst of myoblast fusion but gradually increased thereafter. The localization of transglutaminase underwent a dramatic change from the whole cytoplasm in a diffuse pattern to the cross-striated sarcomeric A band, being strictly colocalized with the myosin thick filaments. When 12-o-tetradecanoyl phorbol acetate was treated to the cultures to induce a sequential disassembly of thin and thick filaments, transglutaminase was strictly colocalized with the myosin thick filaments even in the myosacs, of which most of the thin filaments were disrupted. Moreover, monodansylcadaverine, a competitive inhibitor of transglutaminase, reversibly inhibited the myofibril maturation. In addition, myosin heavy chain was found to be one of the potential substrates for transglutaminase in developing muscle cells. These results suggest that transglutaminase may play a crucial role in myofibrillogenesis of developing chick skeletal muscle.

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Developmental Regulation of Inwardly Rectifying K^+ Channel and Its Possible Role during Myogenesis

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The developmental changes of inwardly rectifying K^+ channel activity in cultured chick muscle cells were examined using the whole-cell and cell-attached configurations of the patch-clamp techniques. In contrast to the outwardly rectifying K^+ currents that were measurable in whole stages of myoblasts differentiation, the inwardly rectifying K^+ currents appeared first only in the aligned myoblasts, but not in the replicating myoblasts, and gradually increased as the development progressed. When the single channel currents of inwardly rectifying K^+ channel in aligned myoblasts were recorded in the cell-attached configuration, unitary I-V relations were rectified and unitary conductance was 21 pS with 140 mM K^+ in the pipet. Application of 0.1 mM Ba^{2+} to the bath abolished up to about 80 % of both the inwardly rectifying K^+ currents and resting membrane potential but not the outwardly rectifying K^+ currents. In conclusion, the inwardly rectifying K^+ channels that express first in the aligned myoblasts are likely to play a role in setting the resting membrane potential during chick myogenesis.