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IPMK and β-catenin take part in PLC-β1-dependent signaling pathway during myogenic differentiation

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Phospholipase C (PLC)-β1 catalytic activity plays an essential role in the initiation of myogenic differentiation but the effectors involved in its signaling pathway are not well defined[1,2]. Here, we show that the overexpression of the Inositol Polyphosphate Multikinase (IPMK) promotes myogenic differentiation, and that IPMK targets the same cyclin D3 promoter region activated by PLC-β1. Moreover, cyclin D3 promoter activation relies upon c-jun binding to the promoter, both in response to PLC-β1 and to IPMK overexpression. Furthermore, both IPMK and PLC-β1 overexpression determines an increase in β-catenin translocation and accumulation to the nuclei of differentiating myoblasts resulting in higher MyoD activation. Therefore, our data show that PLC-β1, IPMK and β-catenin are mediators of the same signaling pathway that regulates cyclin D3 and myosin heavy chain (MYH) induction during myogenic differentiation.

References

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Keywords
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