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Activation and nuclear translocation of PKCε promotes skeletal muscle cell differentiation via HMGA1 down-regulation

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The role of novel PKCs in skeletal muscle differentiation has recently emerged. PKCθ is the most expressed isoform of PKCs in muscle and it promotes the fusion of myoblasts [1]. Recently, we have demonstrated that PKCε is implicated in myocardiocyte differentiation of bone marrow mesenchymal stem cells [2] but the role of PKCε in skeletal muscle cell regeneration has only recently emerged [3].

We here demonstrate that both nuclear and cytoplasmic fractions of PKCε are up-regulated during in vitro C2C12 cell line and satellite cell differentiation. We also show that PKCε is able to modulate myogenic differentiation genes via a down-modulation of HMGA1 proteins that promotes myogenin accumulation and mature myoblast formation. To study the effects of PKCε on muscle regeneration, we have used the in vivo model of CTX-induced skeletal muscle injury. We show that the up-regulation of PKCε also occurs in vivo, particularly in the centro-nucleated regenerating fibers that are derived from the fusion process of the resident satellite cells, suggesting a role for PKCε in human satellite cell-driven muscle repair and substitution, with clinically relevant implications in human muscle pathology.

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Key words
Muscle stem cells, PKCε, skeletal muscle differentiation.

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