MyoD induces ARTD1 and nucleoplasmic poly-ADP-ribosylation during fibroblast to myoblast transdifferentiation

Bisceglie, Lavinia; Hopp, Ann-Katrin; Gunasekera, Kapila; Wright, Roni H; Le Dily, François; Vidal, Enrique; Dall’Agnese, Alessandra; Caputo, Luca; Nicoletti, Chiara; Puri, Pier Lorenzo; Beato, Miguel; Hottiger, Michael O

Abstract: While protein ADP-ribosylation was reported to regulate differentiation and dedifferentiation, it has so far not been studied during transdifferentiation. Here, we found that MyoD-induced transdifferentiation of fibroblasts to myoblasts promotes the expression of the ADP-ribosyltransferase ARTD1. Comprehensive analysis of the genome architecture by Hi-C and RNA-seq analysis during transdifferentiation indicated that ARTD1 locally contributed to A/B compartmentalization and coregulated a subset of MyoD target genes that were however not sufficient to alter transdifferentiation. Surprisingly, the expression of ARTD1 was accompanied by the continuous synthesis of nuclear ADP ribosylation that was neither dependent on the cell cycle nor induced by DNA damage. Conversely to the H2O2-induced ADP-ribosylation, the MyoD-dependent ADP-ribosylation was not associated to chromatin but rather localized to the nucleoplasm. Together, these data describe a MyoD-induced nucleoplasmic ADP-ribosylation that is observed particularly during transdifferentiation and thus potentially expands the plethora of cellular processes associated with ADP-ribosylation.

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Highlights
- MyoD-dependent transdifferentiation of IMR90 to myoblasts induces ARTD1 expression
- Transdifferentiation induces nuclear ARTD1-dependent ADP-ribosylation in myoblasts
- This ADP-ribosylation is induced independent of cell cycle and DNA damage
- ARTD1-mediated poly-ADP-ribosylation localizes to the nucleoplasm in myoblasts

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