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## Is mild hypothermia cool enough to protect cardiac cells after hypoxia? How does propofol affect this effect?

*Soltani P. (1), Wollersheim S. (1), Krauss A. (1), Tong G. (1), Berger F. (1,2), Schmitt K.R.L. (1)*
*German Heart Institute Berlin, Germany (1)*
*Charité - Universitätsmedizin Berlin (2)*

Objectives:
The aim of this study was to elucidate cellular mechanisms responsible for the hypothermia induced protection of cardiac cells from apoptosis after a hypoxic event as observed cardiac arrest.. Hence we investigated the role of therapeutic mild hypothermia (34°C) and propofol on cardiac cells after a hypoxic event.
Methods:
Acute hypoxic conditions were simulated in a cardiac cell line (H9c2) using $CoCl_2$ [30mM] for 1h followed by a treatment with mild hypothermia and/or propofol [50µM] for 24h. Cell survival was determined after 24h of treatment using trypane blue staining. Apoptotic cellular activity was analysed by immunohistochemistry (M30-CytoDEATH) for cleaved keratin 18 as a caspase substrate. Regulation of key effector protein caspase 3 in the intrinsic apoptosis was investigated using western blotting and quantified by densitometry measurements.
Results:
Mild hypothermic treatment (34°C) after hypoxia significantly increased survival of the cardiac cells after 24h. Propofol treatment had no influence on cell survival after hypoxia. The combined application of mild hypothermia and propofol to cardiac cells did not show a significantly higher cell survival than treatment with mild hypothermia alone. Caspase triggered cleavage of keratin 18 occured earlier and more extensive in normothermic cells. In densitometry analyses effector caspase 3 significantly decreased in cardiac cells starting at 6h and continuing after 24h of mild hypothermia.
Conclusions:
Mild hypothermia is efficient enough to protect cardiac cells from apoptosis after a hypoxic event. Both, apoptosis process and execution is effectively reduced by mild hypothermia. Propofol treatment at 50µM after a hypoxic event does not have a significant influence on apoptosis in hypoxic cardiac cells, neither as singular nor as combined treatment with mild hypothermia.