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Racial Differences in ROS Production and SOD Activity Following Induced Inflammation

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Greater hypertension incidence in African Americans (AA) may be due to subclinical vascular dysfunction caused by inflammation and heightened levels of reactive oxygen species (ROS). At rest, AA and Caucasian Americans (CA) exhibit divergent inflammation, ROS production, and ROS clearance both *in vivo* and *in vitro*. However, racial differences in ROS production and clearance following induced inflammation are not fully elucidated. **PURPOSE:** To evaluate racial differences in ROS production and superoxide dismutase (SOD) activity, a major contributor to ROS clearance, in AA and CA human umbilical vein endothelial cells (HUVECs) following induced inflammation. **METHODS:** Following triplicate, parallel experiments with inflammation induced in eight HUVEC cell lines (n=4/group) using tumor necrosis factor-alpha (TNF-, 50ng/ml), cell lysate samples from Control and TNF- treatment were collected at 4 (4H) and 24 hours (24H) post-stimulus. Fluorescence detection was used to quantify ROS production and viable cells alive at 4H and 24H post-stimulus. Control and TNF- cell lysate were subsequently assayed for SOD activity and protein concentration. **RESULTS:** TNF- treatment significantly increased ROS production per living cell in all HUVECs at 24H compared with Control (CellROX/Hoechst ratio- 24H TNF-: 1.44 ± 0.20 , 24H Control: 1.37 ± 0.21 , $p=0.004$). Notably, AA HUVECs did not exhibit a significant higher ROS production at 24H compared with 4H of TNF- treatment (4H: 1.23 ± 0.20 , 24H: 1.40 ± 0.25 , $p=0.08$). However, CA HUVECs exhibited significantly greater ROS production at 24H compared with 4H of TNF- treatment (4H: 1.29 ± 0.13 , 24H: 1.49 ± 0.15 , $p=0.003$). Further, AA HUVECs SOD Activity was similar between conditions and time points, yet CA HUVECs SOD Activity was significantly greater at 24H as compared to 4H in the Control condition with a similar trend in the TNF- condition (SOD Activity (U/mg)- Control 4H: 0.240 ± 0.12 , Control 24H: 0.622 ± 0.17 , $p=0.003$; TNF- 4H: 0.220 ± 0.09 , TNF- 24H: 0.480 ± 0.17 , $p=0.07$). **CONCLUSION:** AA and CA exhibit divergent ROS production and SOD Activity following induced inflammation (*in vitro* model), suggesting higher ROS production in CA and a better ability to buffer ROS production in AA.

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