

TACSM Abstract

Effects of Acute Cold Exposure on Plasma Biomarkers Associated with Cardiovascular Disease

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ABSTRACT

Inflammatory cytokines and lipid mediators are used as biomarkers for CVD risk. Cold exposure has been suggested to improve some of these biomarkers. We measured cardiovascular inflammatory and lipid biomarkers to expand our knowledge of cold exposure and CVD risk. Interleukin-1 Beta (IL-1 B) and Chemokine Ligand 2 (CCL2) are inflammatory cytokines associated with various disease states. Free fatty acids (FFA) are released from fat cells in response to stress. We evaluated the effects of acute 30 min cold exposure on these blood biomarkers. We hypothesized that the inflammatory markers and plasma FFA levels would increase at 2-h post-cold exposure. Twenty subjects (9 females, 23.9 ± 2.7 sd y, 1.71 ± 10.2 sd m, 74.2 ± 13.5 sd kg, 19.4 ± 7.4 sd %BF, 64.5 ± 15.3 sd kg FFM) were subjected to a 30-min seated cold exposure while metabolic data was collected via indirect calorimetry. Shivering started immediately upon cold exposure and ceased within 10 seconds following cold exposure. Estimated resting energy expenditure (kcal/min) during the exposure period (1.73 ± 0.7 sd, 1.47 ± 0.6 sd, 1.36 ± 1.0 sd for min 5, 15, and 30, respectively) was two-fold higher than pre-cold. Venous plasma was collected at pre-cold period, immediately after cold exposure, and 2 h post-cold, centrifuged, and stored at -80°C for subsequent biomarker analysis. ELISAs were used to measure plasma inflammatory cytokines; interleukin-1 beta (IL-1 B) and chemokine ligand 2 (CCL2) biomarkers and plasma free fatty acid (FFA) during and following cold exposure. Pre-cold IL-1B (pg/ml), CCL2 (pg/ml), and FFA (mM) values were 19 ± 3.6 se, 2.4 ± 0.7 se, and 507 ± 87 se, respectively. Immediate post-cold values were 18 ± 3.8 se, 3.4 ± 1.2 se, and 412 ± 42 se, respectively. Finally, 2 h post-cold values were 20 ± 3.6 se, 2.6 ± 0.7 se, and 458 ± 48 se, respectively. There was a 3.4 ± 0.7 se ($p < 0.05$) increase in IL-1 B plasma levels immediately after cold exposure that lasted up to two hours. CCL2 plasma levels and FFA were not different from baseline during the post-cold period. We conclude that acute cold exposure may worsen CVD risk through a select inflammatory response. Additional analyses of our samples will expand the possible list of affected CVD risk biomarkers. Whether or not extended exposure to cold would exacerbate these marker levels or affect the other markers measured is not known.