FIRE SIMULATION EXPOSURE CAUSES IMPAIRMENT OF ENDOTHELIAL FUNCTION AND INCREASED THROMBOGENICITY IN HEALTHY FIREFIGHTERS

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Background: The risk of acute myocardial infarction in firefighters is increased during fire suppression duties, and is likely to reflect a combination of factors including extreme physical exertion and heat exposure. We assess the effects of physical exertion and heat exposure on vascular function and thrombosis in firefighters.

Methods: In an open label randomized cross-over study, seventeen healthy, non-smoking firefighters (age 41±7 years, 15 male) performed a standardized training exercise in a fire simulation facility (average temperature 400°C) or a sham exposure for 20mins. Bilateral forearm blood flow was measured before and during unilateral intra-brachial infusion of acetylcholine (5 - 20 µg/min), sodium nitroprusside (2 - 8 µg/min) and verapamil (10 - 100 µg/min) 2-4 hours after exposure. Thrombus formation and platelet activation was measured using the Badimon chamber at 2 hrs and by flow cytometry of whole blood immediately following and 24 hrs after each exposure.

Results: Following fire simulation training, core temperature in firefighters increased by 1.2 ± 0.48 °C and weight was reduced by 0.46 ± 0.14kg (P<0.001 for both). Intra-arterial infusions increased forearm blood flow in a dose dependent manner (P<0.001), but vasodilatation was impaired following exposure to fire simulation in response to acetylcholine (P=0.01) and sodium nitroprusside (P=0.004), but not verapamil (P=0.38). Exposure to fire simulation training increased thrombus formation under low- and high shear conditions by 60% (change in thrombus area 5,038 µm², 95% CI 3,006-7,009µm²) and 58% (change in thrombus area 5,582µm², 95% CI 1,795-9369µm²) respectively. This was associated with an increase in platelet-monocyte binding of 21%(absolute change 7%, 95%CI 0.8-13%, P=0.03) from baseline following fire simulation training.

Conclusions: Exposure to extreme heat and physical exertion during fire suppression impairs vascular function and increases thrombus formation in healthy firefighters. Our findings suggest pathogenic mechanisms to explain the association between fire suppression activity and acute myocardial infarction in firefighters.