

modelling. **RESULTS:** Pilots/aircrew had higher plasma levels of inflammatory mediators compared to controls, notably CCL2 (134.5 vs 124, 99% posterior probability [pp]) MPO (19.6×10^3 vs 16.3×10^3 , 92% pp) and TNF- α (6.4 vs 5.8, 87.2% pp). Conversely, CRP (11.5×10^5 vs 9.7×10^5 , 92% pp), and IL6R (5.1×10^4 vs 4.8×10^4 , 87% pp) were lower in pilots. TC stimulation elicited greater inflammatory reactivity in pilots vs controls, as TNF- α , IL-6, IL-1b, CCL-2, CCL8, IL-8 were all higher in response to LPS-stimulation. **CONCLUSIONS:** The distinct profile of peripheral immuno-inflammatory biomarker expression and reactivity suggests a link between WMH and inflammatory activation.

Learning Objectives

1. The audience will learn about the methods used to evaluate for dysfunctional central and peripheral immuno-inflammatory activation.
2. The audience will learn about a distinct profile of peripheral immuno-inflammatory biomarker expression and reactivity suggesting a link between WMH and inflammatory activation in a sample of RCAF fighter pilots and jumpers.

Monday, 05/22/2023
Napoleon Ballroom C1-C2

10:30 AM

[S-07]: SLIDES: DO YOU REALLY WANT TO GO THAT HIGH? BAROTRAUMA & DCS

Chair: William Buck Dodson
Co-Chair: Bria Morse

[33] DECOMPRESSION SICKNESS RISK ASSOCIATED WITH REPEAT ALTITUDE EXPOSURE

Vivienne Lee¹, Desmond Connolly², Timothy D'Oyly¹, Thomas Smith²

¹QinetiQ PLC, Farnborough, United Kingdom; ²King's College London, London, United Kingdom

(Original Research)

INTRODUCTION: In 2017, two Royal Air Force parachute jump instructors experienced symptoms of severe decompression sickness (DCS) whilst undertaking despatcher duties at 25,000 ft. This prompted more conservative altitude exposure limitations and denitrogenation requirements for high altitude parachuting. Despatchers' risk of DCS is greater than aircrew and parachutists due to greater physical activity during cabin decompression, but absolute risk is uncertain due to lack of representative research. This study investigated the risk to despatchers following the new procedures and explored the potential for safely conducting repeat exposures in a single duty period. **METHOD:** Fifteen men aged 20 to 50 yr, without 'right-to-left' vascular shunts, underwent repeat altitude chamber decompression breathing 100% oxygen. Phase 1 comprised two ascents to 25,000 ft, 1 hr followed by 1.5 hr, each with 1 hr denitrogenation at 15,000 ft. In Phase 2, an identical initial ascent was followed by two 1.5 hr ascents to 22,000 ft with 30 min denitrogenation at 15,000 ft. All ascents were separated by 1 hr breathing air at ground level. Participants undertook activities representative of parachutist despatchers throughout. Cardiac echocardiography was undertaken every 15 mins to monitor venous gas emboli (VGE) loads. Participants diagnosed with DCS were recompressed and did not proceed to further ascents of that phase. **RESULTS:** Four cases of DCS were diagnosed from 29 initial ascents to 25,000 ft. One participant was diagnosed with DCS during subsequent ascent to 25,000 ft. No DCS occurred at 22,000 ft. During initial exposures of both phases, the majority of participants produced heavy VGE loads, from multiple limbs, within 30 mins. Participants tended to exhibit lighter, and later, VGE loads during subsequent exposures. Older participants (>40 yr) were more likely to experience symptoms and early heavy VGE loads. **CONCLUSIONS:** Exposure to 25,000 ft for 1 hr, with exercise, presents a risk of DCS. DCS is more likely during an initial ascent to 25,000 ft compared to a second ascent occurring after about an hour.

VGE loads tend to be reduced in subsequent ascents indicating carryover benefit of denitrogenation from prior ascents. Individuals over 40 yr are at greater risk of DCS.

Learning Objectives

1. The presentation will discuss the factors influencing risk of decompression sickness with repeated (same day) exposure to a provocative altitude. The audience should keep in mind the following: altitude; duration; exertion; denitrogenation; time spent breathing 100% oxygen; age; exposure interval.
2. The audience will be familiarised with the nature of venous gas emboli (VGE) loads with exertional decompression stress, emphasizing early onset, heavy and persistent bubble loads despite effective denitrogenation procedures.

[34] EARLY PATHOPHYSIOLOGICAL RESPONSES TO EXERTIONAL, NON-HYPOXIC, HYPOBARIC DECOMPRESSION STRESS

Desmond Connolly¹, Leigh Madden², Stephen Harridge³, Victoria Edwards¹, Timothy D'Oyly¹, Vivienne Lee¹

¹QinetiQ PLC, Farnborough, United Kingdom; ²University of Hull, Hull, United Kingdom; ³King's College London, London, United Kingdom

(Original Research)

INTRODUCTION: Consistent blood biomarkers of hypobaric decompression stress remain elusive. Laboratory investigation of decompression sickness (DCS) risk with repeat (same-day) exposure to 25,000 ft pressure altitude enabled investigation of pathophysiological responses to exertional decompression stress. **METHODS:** Fifteen healthy men, aged 20 to 50 yr, undertook two ascents to 25,000 ft, for 60 and 90 min, breathing 100% oxygen, each following an hour of denitrogenation. An hour separated the ascents, breathing air at 400 ft amsl. Venous blood was sampled pre-exposure (T0), after ascent two (T8) and next morning (T24). Besides whole blood hematology, endothelial microparticles (EMPs) were analyzed by flow cytometry, and selected proteins by enzyme-linked immunosorbent assay (ELISA). Targets included cytokines, markers of endothelial function, inflammation, coagulopathy, oxidative stress, brain insult, cortisol and creatine kinase. Blood/plasma volume shifts and diurnal variation were accounted for. **RESULTS:** Participants experienced heavy venous gas emboli (VGE) loads with three exposures curtailed due to limb bend DCS. Acute (T8) hematological effects on neutrophils (mean 72% increase), eosinophils (40% decrease), and monocytes (37% increase) normalized by T24. Mean five-fold elevation of interleukin-6 (IL-6) at T8 ($P < 0.00001$) was pro-inflammatory (suppression of IL-10 and absent cortisol stress response). Complement system activation increased peptide C5a ($P < 0.05$), and mean C-reactive protein (CRP) rose by 100% over baseline ($P < 0.005$), supporting an acute phase response. Increased circulating total EMPs and tissue factor (TF) support endothelial dysfunction and oxidative stress influenced enzymatic and non-enzymatic markers. Glial fibrillary acidic protein (GFAP), a sensitive brain injury marker, increased 10% at T24 ($P = 0.015$), and T8 serum levels of the neurotransmitter glutamate tended to rise ($P = 0.078$). **DISCUSSION:** Pulmonary VGE loading appears to drive IL-6 release from neutrophils and/or endothelial cells, determining the magnitude of the acute phase response (CRP). Hematological responses and IL-6 normalized quickly but increased CRP, C5a, TF, total EMPs, GFAP and neutrophil gelatinase-associated lipocalin (indicating neutrophil activation) persisted, suggesting ongoing susceptibility to further decompression stress. The GFAP and glutamate data warrant concern; potential brain markers of decompression stress require further evaluation.

Learning Objectives

1. The presentation will outline the early blood biomarker responses to exertional decompression stress, emphasizing hematological, cytokine and acute phase (inflammatory) responses and suggesting a likely pulmonary basis for these in relation to oxidative stress and impact of venous gas emboli.