Results A total of 2150 echocardiograms were analysed over the 18 months. The median age of our cohort was 69 years (22 - 94). 66% of our patients were males and 34% females. 146 patients were found to have aortic dilatation. Therefore, the incidence of aortic dilatation was 6.8% in our study population.

Conclusion The incidence of aortic dilatation in our hospital population of 6.8% was significantly higher than we expected. It is a staggering 1000 fold increase when compared to current literature surrounding the incidence of aortic aneurysm which is the possible end point of aortic dilatation[1,2]. Based on the incidence established in this study, our hospital alone would have at least 400 patients with a dilated aorta in a year. Due to the potential detrimental prognosis of aortic dilatation, further investigations are certainly warranted to identify risk factors related to the development and progression of aortic dilatation as well as the pattern of progression.

REFERENCES

Conflict of Interest Nil
monitoring, CT coronary angiography and cardiac MRI. Athletes with risk factors for coronary artery disease (CAD) were excluded. A HRE was defined as a peak systolic blood pressure during CPET of ≥220mmHg in males and ≥190mmHg in females. Data were analysed to evaluate for a significant relationship between the HRE and the presence of pathological coronary calcification (calcium score >70th Centile), myocardial fibrosis and ventricular tachycardia on 24hour Holter monitoring.

Over a third of athletes (36.8%) compared to only 7.6% of controls exhibited a HRE on CPET testing (p<0.001). Athletes with a HRE did not differ in age (median 52), sex distribution, number of years of endurance training or hours of training per week compared to athletes without a HRE. Of the athletes with a HRE, 17.8% had pathological coronary calcification compared to 19.8% of athletes without (p=0.83), 12.7% had pathological myocardial fibrosis compared to 9.3% without (p=0.59) and 11% had non-sustained VT compared to 5.4% without (p=0.21).

A hypertensive response to exercise is highly prevalent in veteran endurance athletes but present in only a minority of sedentary controls. The HRE, measured according to current methodology however, was not predictive for the presence of pathological coronary calcification, myocardial fibrosis or ventricular arrhythmias in this athlete cohort. Studies thus far are yet to explain the increased prevalence of such findings amongst veteran athletes or determine if such findings correspond to adverse cardiovascular events. Future studies should address these issues in order that evidence-based guidance for risk stratification and pre-participation screening of master athletes can be developed.

Conflict of Interest Nil

132 INVESTIGATING THE LOWEST THRESHOLD OF VASCULAR BENEFITS FROM LDL LOWERING WITH A PCSK9 INHIBITOR IN HEALTHY VOLUNTEERS – RESULTS FROM THE INTENSITY-LOW STUDY

Paul Cacciotto, Michalis S Kostapanos, Holly Pawey, Annette Hubsch, Ian B Wilkinson, Joseph Cheriyan, University of Cambridge; Cambridge University Hospitals NHS Foundation Trust

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Background There is a continuous relationship between low density lipoprotein cholesterol (LDL-C) and cardiovascular risk. The cardiovascular benefits of reducing LDL-C in low risk normocholesterolaemic subjects is unknown. The INTENSITY LOW study sought to determine whether lowering LDL-C by alirocumab improves endothelial function in healthy individuals.

Method This was a single-centre, randomised, single-blind, placebo-controlled study.

30 healthy normocholesterolaemic participants aged 18-45 were randomized 1:1 to receive either alirocumab 150mg or placebo subcutaneously. Forearm blood flow (FBF) as measured by venous occlusion plethysmography was undertaken at baseline and again after 2 weeks therapy. Endothelium-dependent and -independent vasodilation were assessed during intrararterial infusion of acetylcholine (ACh) and sodium nitroprusside (SNP) respectively. All participants then received a further dose of their original treatment allocation, as well as atorvastatin 20mg once daily, and measurements were undertaken after a further 2 weeks. Pulse wave velocity (PWV), Augmentation Index (Alx) and carotid Intima-media thickness (CIMT), as well as LDL-C levels, were measured at each timepoint.

Results Alirocumab reduced LDL-C compared to placebo by 48% (ΔLDL-C -1.00mmol/L, 95% CI 0.89-1.11, P<0.001). There was no difference in endothelium-dependent forearm responses to ACh between alirocumab and placebo (ΔFBF response 5.13 ml(100ml)-1min-1, 95% CI 4.07 -6.19, P=0.977). However, forearm ACh responses improved when comparing alirocumab + atorvastatin to atorvastatin alone (ΔFBF response 8.06 ml(100ml)-1min-1, 95% CI 7.06-9.06, P=0.01). There was no significant change in endothelium-independent responses to SNP when comparing alirocumab to placebo (ΔFBF response 5.85ml(100ml)-1min-1, 95% CI 4.88 – 6.82, P=0.462), or alirocumab + atorvastatin to atorvastatin alone (ΔFBF response 6.00ml(100ml)-1min-1, 95% CI 5.07 – 6.93, P=0.389). There was no significant change in PWV with alirocumab therapy (ΔPWV -0.271 ± 0.223 ms-1, P=0.271), or with the addition of statin to alirocumab therapy (ΔPWV 0.073 ± 0.23ms-1, P=0.753). There was no significant change in Alx with alirocumab therapy (ΔAlx -1.143 ± 2.884, p=0.692) or with the addition of statin to alirocumab therapy (ΔAlx -4.203 ± 4.102, p=0.306). CIMT was reduced with alirocumab therapy (ΔCIMT -0.051 ± 0.026mm, p=0.05), but there was no significant change with addition of statin to alirocumab therapy (ΔCIMT -0.005 ± 0.031mm, p=0.882).

Conclusion Alirocumab significantly reduced LDL-C compared to placebo without any change to endothelial function, as measured by forearm plethysmography, or to markers of arterial stiffness, as measured by PWV and Alx, in normocholesterolaemic healthy subjects. However, an improvement in