expression *in vivo*, although there were significant inter-patient variations. There was a significant reduction in circulating T cells following CMR.

**Conclusion** CMR was not associated with DNA damage *in vivo*. γ-H2AX expression varied markedly between individuals, therefore small studies using γ-H2AX as a marker of DNA damage should be interpreted with caution. CMR was associated with a statistically significant reduction in viable leukocytes, although the clinical relevance of the magnitude is unclear. Further work is warranted to contextualise these findings and delineate their impact.

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**010** GENERATION OF A FORMULA FOR CAROTID-FEMORAL PATHLENGTH DETERMINATION FOR USE IN PWV ASSESSMENT

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10.1136/heartjnl-2017-311399.10

**Background** Aortic arterial stiffening is an independent predictor for future cardiovascular events. Calculation of Carotid-Femoral Pulse Wave Velocity (cPWV) is currently the clinical gold standard for measuring arteriosclerosis, with cPWV now recommended by the European Society of Cardiology for the guidance of initiating preventative treatments. Despite this, no consensus exists on how best to obtain the pathlength component of the calculation with large intercentre variation in how this is performed. The aim of the current study was to generate a calculation to produce a standardised pathlength that can be generated from easily obtainable clinical measures.

**Methods** 1183 participants free from cardiovascular disease (CVD) underwent whole body MRI as part of the TASC-FORCE study. The distance between the carotid and femoral vessels was obtained by tracing the arterial centreline between the two. Backward linear regression was then used to generate a formula for calculating pathlengths based on easily obtainable clinical metrics. This calculation was then validated in an external cohort of 128 individuals with and without CVD who had also undergone MRI.

**Results** Various allometric and cardiovascular values were included in the analysis. Carotid-femoral pathlength could be calculated as follows:

\[
\text{Distance} = 100.36 + (0.70 \times \text{Age [years]}) + (137.89 \times \text{Height [m]}) + (0.52 \times \text{Weight [kg]}) - (0.17 \times \text{Pulse}) + (46.16 \text{ [female], 54.32 [male]})
\]

When compared with the actual measured distance in the original cohort this differed by −0.05 mm SD +28.5 mm, p=0.962 for difference. When this formula was then applied in the external validation cohort there was a small overestimation by 10.07±25 mm (p>0.001). This is performed. The aim of the current study was to generate a formula for calculating pathlengths based on easily obtainable clinical metrics. This calculation was then validated in an external cohort of 128 individuals with and without CVD who had also undergone MRI.

**Conclusion** Using simple allometric measures, carotid-femoral pathlength can be calculated with good accuracy. This holds promise for improving interstudy and intercentre reproducibility, thus expanding the utility and applicability of PWV calculation in clinical practice. In future, the predictive ability of the formula can be tested in disease discrimination cohorts to further assess its clinical applicability.