kgAT) (+2.44 ml/kg/min [CI 0.6,4.2], $p=0.009$), time to AT (+115s [CI 54.3,175.9], $p<0.001$) and exercise time (max ET) (+108s [CI 33.7,182.2], $p=0.005$). The exercise group also demonstrated greater reduction in systolic BP (-7.3 mmHg [CI -11.7,-2.8], $p=0.002$), BMI (-0.8 kg/m² [CI-1.1,-0.4], $p<0.001$), anxiety (-2.6 [CI-3.6,-1.6], $p\leq 0.001$) and depression (-1.1 [CI -2.0,-0.2], $p=0.015$) scores. At T6m patient reported exercise adherence was comparable to baseline PA, in 33/34 of the exercise group attending for follow up. Most exercise gains dissipated with the exception of time to AT ($p=0.002$), max ET ($p=0.003$), VO₂/kgAT ($p=0.04$) and anxiety score ($p<0.001$). There were no sustained episodes of atrial or ventricular arrhythmias. The incidence of NSVT did not differ between time points ($p=0.09$).

Conclusion A 12-week HIT programme in young patients with HCM offers considerable gains in fitness and psychological outcomes, with no increase in arrhythmic burden. Further research is still required to assess the long-term safety of high intensity exercise in the HCM population. At T6m exercise levels as well as most physiological adaptations and health benefits returned to baseline, as seen in other studies when formal participation in an exercise programme comes to an end. This highlights the importance of the implementation of strategies to encourage ongoing engagement in PA. Potential solutions include identification of barriers to exercise, as well as adoption of novel tele-rehabilitation approaches.

C IMPAIRED MYOCARDIAL ENERGETICS AS THE BASIS FOR EXERCISE-INDUCED PULMONARY CONGESTION IN HEART FAILURE WITH PRESERVED EJECTION FRACTION

¹Matthew K Burrage*, ¹Moritz Hundertmark, ²Jack J Miller, ¹Andrew JM Lewis*, ¹Oliver J Rider*, ¹University of Oxford Centre for Clinical Magnetic Resonance Research, Radcliffe Department of Medicine, University of Oxford, OX3 9DU, UK; ²Department of Physics, Clarendon Laboratory, University of Oxford, OX1 3PU, UK; *contributed equally as senior authors

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Background Abnormal cardiac mitochondrial function and energetics are implicated in the pathogenesis of heart failure with preserved ejection fraction (HFpEF). Transient pulmonary congestion during exercise is emerging as an important determinant of reduced exercise capacity and symptoms in HFpEF. We sought to determine if an abnormal cardiac energetic state underpins this process.

Methods We recruited 42 patients across the spectrum of diastolic dysfunction and HFpEF (healthy controls $n=10$, type 2 diabetes (T2DM) $n=9$, HFpEF $n=14$, and severe diastolic dysfunction due to cardiac amyloidosis $n=9$). Cardiac energetics were measured using phosphorus spectroscopy to define the myocardial phosphocreatine to adenosine triphosphate ratio (PCr/ATP). Cardiac function was assessed by cardiovascular magnetic resonance (CMR) cine imaging and echocardiography, and pulmonary congestion using MR proton density mapping. Studies were performed at rest and during submaximal exercise using an MRI-ergometer.

Results Paralleling the stepwise decline in diastolic function across the groups (E/e' ratio $p<0.001$) was an increase in NT-pro BNP ($p<0.001$) and a reduction in PCr/ATP (control 2.00 [1.86, 2.15], T2DM 1.71 [1.61, 1.91], HFpEF 1.66 [1.44, 1.89], cardiac amyloidosis 1.30 [1.16, 1.53], $p<0.001$). During 20W exercise, reduced left ventricular (LV) diastolic filling rate ($r=0.41$, $p=0.008$), left atrial (LA) dilatation ($r=-$

0.35, $p=0.03$), reduced right ventricular (RV) contractile reserve (RV ejection fraction change $r=0.46$, $p=0.003$), reduced right ventricular-pulmonary arterial (RV-PA) coupling ($r=0.36$, $p=0.02$) and right atrial dilatation ($r=-0.68$, $p<0.001$) were all linked to this reduction in PCr/ATP. Along with these changes, proton-density mapping revealed transient pulmonary congestion in patients with HFpEF (+4.4% [0.5, 6.4]) and cardiac amyloidosis (+6.4% [3.3, 10]), which was not seen in healthy controls (0.25% [-1.8, 3.1]) or T2DM without HFpEF (0.8% [-1.7, 1.9]). Importantly, the development of exercise-induced pulmonary congestion was associated with reduced PCr/ATP ($r=-0.36$, $p=0.02$).

Conclusions A gradient of myocardial energetic deficit exists across the spectrum of HFpEF. This energetic deficit is related to markedly abnormal exercise responses in all four cardiac chambers, which leads to detectable pulmonary congestion. The findings support an energetic basis for transient exercise-induced pulmonary congestion in HFpEF.

D RADIOTRANSCRIPTOMIC ANALYSIS OF PERIVASCULAR ADIPOSE TISSUE QUANTIFIES VASCULAR INFLAMMATION IN COVID-19 FROM ROUTINE CT ANGIOGRAMS: STRATIFICATION OF "NEW UK VARIANT" INFECTION AND PREDICTION OF IN-HOSPITAL OUTCOMES

¹Christos P Kotanidis*, ¹Cheng Xie, ²David Adlam, ³Jonathan CL Rodrigues, ^{1,4}Muhammad Siddique, ⁵Helen Lockstone, ^{1,6}Sheena Thomas, ¹Rafail Kotronias, ^{1,7}Evangelos K Oikonomou, ⁸Sheila F Lumley, ¹Stefan Neubauer, ⁹David E Newby, ^{1,10}Keith M Channon, ¹¹John Deanfield, ^{1,6}Charalambos Antoniades. ¹Division of Cardiovascular Medicine, Radcliffe Department of Medicine, University of Oxford, UK; ²Department of Cardiovascular Sciences and NIHR Leicester Biomedical Research Centre, University of Leicester, UK; ³Department of Radiology, Royal United Hospitals Bath NHS Foundation Trust, UK; ⁴Caristo Diagnostics, Oxford, UK; ⁵Wellcome Trust Centre for Human Genetics, University of Oxford, UK; ⁶Acute Vascular Imaging Centre, NIHR Biomedical Research Centre, John Radcliffe Hospital, Oxford University Hospitals NHS Foundation Trust and University of Oxford, UK; ⁷Department of Internal Medicine, Yale-New Haven Hospital, Yale School of Medicine, New Haven, USA; ⁸Nuffield Department of Medicine, University of Oxford, UK; ⁹British Heart Foundation Centre for Cardiovascular Science, University of Edinburgh, UK; ¹⁰British Heart Foundation-National Institute of Health Research Cardiovascular Partnership; ¹¹Institute of Cardiovascular Sciences, University College London, London, UK

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Background Evidence suggests that adverse outcomes in COVID-19 may be driven by a cytokine-induced vascular inflammatory response, caused by SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2).

Aim We aimed to develop a non-invasive method for quantifying cytokine-driven vascular inflammation in patients with acute COVID-19 infection that could allow risk stratification.

Methods We developed a platform for rapid development of novel imaging biomarkers of vascular inflammation, by applying quantitative radiotranscriptomics to images from standard Computed Tomography Angiography (CTA). We used this platform to train a radiotranscriptomic signature (C19-RS) from the perivascular space around the aorta and the internal mammary artery, visualized in routine chest CTAs, to best describe cytokine-driven vascular inflammation, defined using transcriptomic profiles from RNA sequencing data from human arterial biopsies. This signature was tested externally in 435 clinically indicated CT pulmonary angiograms (CTPAs) from patients with or without COVID-19 from 3 different geographical regions.