**Introduction** Peroxisome proliferator-activated receptors (PPARα, β/δ, γ) are important regulators of cardiac fatty acid metabolism. PPARα has the highest binding affinity for fatty acids and is the dominant cardiac PPAR isoform.

The PPARα knockout mouse has been extensively used to investigate the role of PPARα in metabolism and disease. This study aimed to understand the role of PPARα in the aging heart, through assessment of the metabolism of [1–13C] pyruvate, using hyperpolarisation by dynamic nuclear polarisation (DNP).

**Methods** *Animals*- Six young (3–4 months) and six old (20–22 months) controls (129SvEv) and five young (3–4 months) and six old (20–22 months) PPARα-KO mice (on a 129SvEv background) received a hyperpolarised scan, performed between 7 am and 11 am, while mice were in the fed state.

**Hyperpolarised 13C MRS Protocol** [1–13C] pyruvate was hyperpolarised and dissolved as previously described. An aliquot of 0.15 ml of 80 mM hyperpolarised [1–13C] pyruvate solution was injected over 10 s via a tail vein catheter into an anaesthetised mouse, positioned in a 7 T MR scanner. Spectra were acquired for 1 min following injection, with 1 s temporal resolution, using a 15° RF excitation pulse. Signal was localised to the heart using a home-built 13C RF surface coil. Quantified peak areas were input into a kinetic model, described by Atherton et al, and plotted against time. The rate of exchange of the 13C label between pyruvate and its metabolites was termed 13C label incorporation. 13C label incorporation into the bicarbonate pool has been used as a measure of pyruvate dehydrogenase (PDH) flux in the rat.

**Biochemical analysis** Protein levels of glucose transporter (GLUT) 4, medium chain acyl-CoA dehydrogenase (MCAD), PDH kinase (PDK) 2 and PDK4 were measured in cardiac tissue by western blotting. PPARα mRNA levels were assessed using qRT-PCR.

**Results** Young and old PPARα-KO hearts had significantly decreased PPARα mRNA levels relative to controls and a 59% decrease in protein levels of the PPARα target gene, MCAD. Cardiac PDH flux did not differ between young control and PPARα-KO mice, but in old PPARα-KO mice, PDH flux was increased by 96% relative to controls. Analysis of cardiac metabolic proteins revealed a 141% increase in GLUT4 expression in old PPARα-KO mice. There were no differences in PDK2 or PDK4 protein levels, suggesting that the increased PDH flux in aged PPARα-KO mice was not mediated by altered PDH phosphorylation.

**Conclusions** Aged PPARα-KO mice exhibited increased cardiac PDH flux relative to young and old control mice. This may be mediated by increased GLUT4 protein levels, which would facilitate glucose uptake and glycolysis. No changes in PDK2 and PDK4 expression were observed, although the enzymatic activities of these proteins were not examined.

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