ever, given differing risk thresholds in the US and uncertainty about how to integrate Coronary Calcium Scoring (CCS), their optimal management is not yet established. We assessed the role of CCS at lower thresholds in an Australian context.

**Methods:** 1048 participants aged between 40 and 70 (total cholesterol 5.6 mmol/L, 62% female) with a family history of premature CAD were recruited from the community in Melbourne and Hobart. Predicted risk was calculated using the Australian Absolute CVD Risk calculator. CT coronary calcium scoring (CCS) was undertaken in pts at intermediate risk by ACC/AHA criteria (defined as 10-yr 4-15%).

**Results:** Intermediate risk by US guidelines was present in 80%, 571 participants met inclusion criteria for CCS. 285 (22%) and 30% of total and CT screened participants respectively had CCS >0, with a median CCS of 50 (IQR 12–153). In the subgroup undergoing CCS, scores could guide therapy in all patients aged >60 and in intermediate risk patients aged 50–59. Adopting US thresholds would increase positive CCS scans by 75%, though the yield of positive scans falls to a third in those <60 yo.

**Conclusion:** Addition of CCS to lower intermediate risk thresholds can identify a third of pts <50 currently ineligible who may benefit from statins. CAD correlates with age even at lower risk and CCS can guide therapy in over 60% of >60 at intermediate risk.

<table>
<thead>
<tr>
<th>Age/CCS</th>
<th>Low Risk (%)</th>
<th>Intermediate Risk (%)</th>
<th>High Risk (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &lt;50</td>
<td>99 (79%)</td>
<td>51 (89%)</td>
<td>30 (50%)</td>
</tr>
<tr>
<td>Age 50–59</td>
<td>29 (35%)</td>
<td>55 (26%)</td>
<td>41 (50%)</td>
</tr>
<tr>
<td>Age &gt;60</td>
<td>23 (110)</td>
<td>124</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CCS &gt;0 (%)</th>
<th>Low Risk</th>
<th>Intermediate Risk</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &lt;50</td>
<td>95 (79%)</td>
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<tr>
<td>Age &gt;60</td>
<td>130</td>
<td>124</td>
<td></td>
</tr>
</tbody>
</table>

**P** values for differences in positive scan rates by age group are 0.046 (Age <50 vs. Age 50–59) and 0.001 (Age <50 vs. Age >60).

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### S369

#### High-density Lipoproteins Rescue Diabetes-impaired Angiogenesis by Reprogramming Cardiac Metabolic Response to Hypoxia

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High-density lipoproteins (HDL) rescue hypoxia-induced angiogenesis in diabetes, however, the underlying mechanisms remain unknown. Endothelial cell (EC) metabolic reprogramming is a central component of inducing angiogenesis at a site of ischemia. Pyruvate dehydrogenase kinase 4 (PDK4) suppresses oxidative cell metabolism in hypoxia to decrease oxygen consumption and preserve cell survival, but diabetes impairs this adaptation and the angiogenic response. PDK4 knockdown impaired human coronary artery endothelial cell (HCAEC) tubulogenesis in hypoxia by 82% versus controls (*P* < 0.0001). HCAECs were treated with HDL (20µM) or PBS (vehicle) and exposed to glucose (5 or 25mM, 72h), then hypoxia (1.2% O2, 6 h). PDK4 expression was increased by 60% in hypoxia versus the normoxia control (*P* < 0.05). Contrastingly, in high glucose PDK4 expression failed to increase in response to hypoxia. Incubation with rHDL rescued this and elevated PDK4 expression by 40% in hypoxia and high glucose (*P* < 0.01). In parallel, rHDL rescued high glucose-impaired tubulogenesis in hypoxia by 64% versus the PBS/normoxia control (*P* < 0.001). In a murine model of diabetic wound healing, topical application of rHDL (50µg/wound/day) increased the presence of wound CD31+ neovessels by 46%. This supported an increased rate of wound closure in diabetic animals of 30% (*P* < 0.05). rHDL treated wounds from diabetic mice had a striking increase in PDK4 gene (180%) and protein expression (300%) in the early-mid stages post-wounding (*P* < 0.05).

rHDL rescues diabetes-impaired expression of metabolic regulator PDK4, which plays a critical role in EC angiogenesis in hypoxia/ischemia. This has implications for improving cardiovascular outcomes for diabetic patients following myocardial infarction.

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### S40

#### High-intensity Interval Training for Patients with Coronary Artery Disease: Finding the Optimal Balance

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**Background:** There is growing interest in the application of high-intensity interval training (HIIT) for patients with coronary artery disease (CAD) within cardiac rehabilitation (CR), based on the now-robust evidence of the efficacy of HIIT compared to moderate-intensity continuous training (MICT). However, the optimal characteristics of HIIT for optimizing efficacy and patient enjoyment while maintaining patient safety is still unclear. This study aimed to assess a novel HIIT protocol in patients with CAD within CR.

**Methods:** Twenty-one patients with CAD completed 6-weeks (+2 sessions per week) of HIIT within outpatient (phase 2) CR. HIIT comprised 15 repetitions × 30-seconds cycling at ∼85–90% maximum heart rate, interspersed with 30-seconds of active recovery. Training programme progression was individualised based on heart rate and rating of perceived exertion (RPE). Outcome measures included patient safety (exercise-related adverse events), efficacy (peak aerobic capacity, body composition, blood pressure and vascular function) and patient adherence and enjoyment.