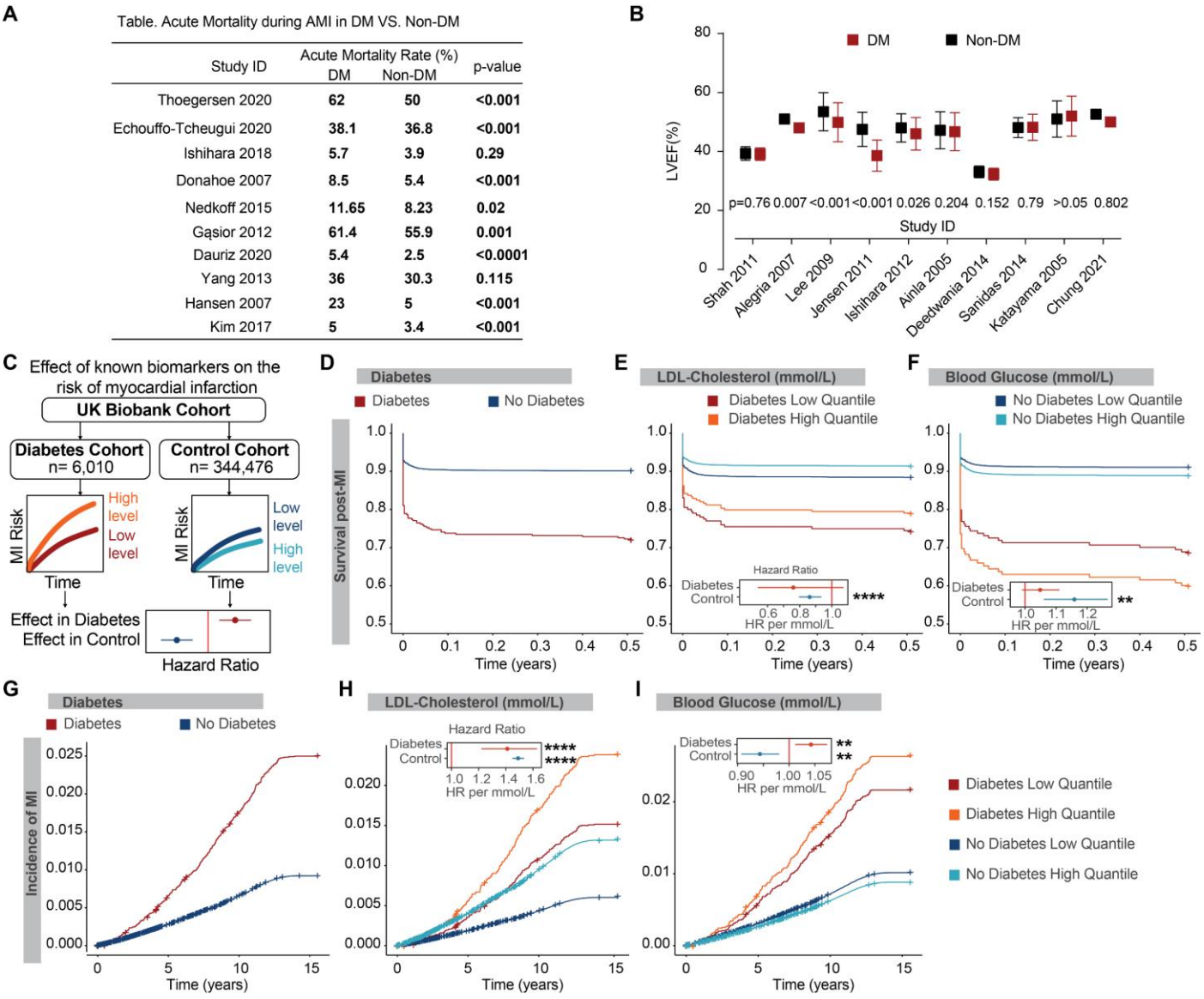
# SUPPLEMENTAL FIGURES AND LEGENDS

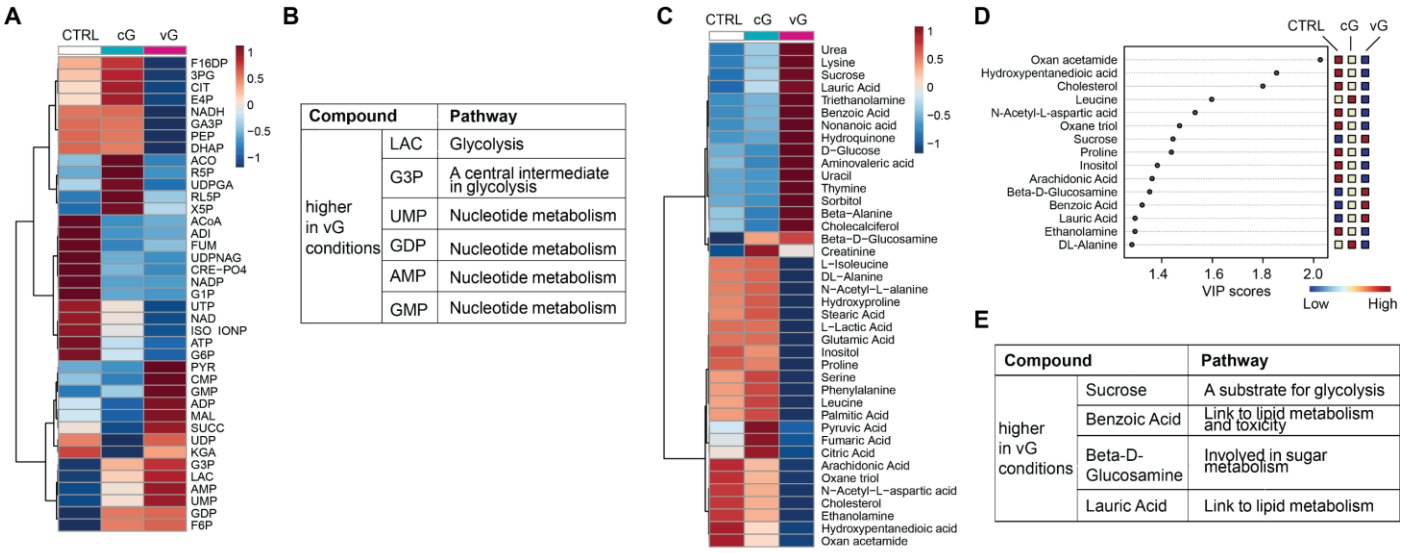
**A**

Table. Acute Mortality during AMI in DM VS. Non-DM

| Study ID | Acute Mortality Rate (%) DM | Non-DM | p-value |
|---|---|---|---|
| Thoegersen 2020 | **62** | **50** | **<0.001** |
| Echouffo-Tcheugui 2020 | **38.1** | **36.8** | **<0.001** |
| Ishihara 2018 | **5.7** | **3.9** | **0.29** |
| Donahoe 2007 | **8.5** | **5.4** | **<0.001** |
| Nedkoff 2015 | **11.65** | **8.23** | **0.02** |
| Gąsior 2012 | **61.4** | **55.9** | **0.001** |
| Dauriz 2020 | **5.4** | **2.5** | **<0.0001** |
| Yang 2013 | **36** | **30.3** | **0.115** |
| Hansen 2007 | **23** | **5** | **<0.001** |
| Kim 2017 | **5** | **3.4** | **<0.001** |




**Supplemental Figure 1. The risk of MI and mortality from MI in DM and non-DM individuals in clinical, epidemiological data and in the UK Biobank Cohort.** (**A**) Clinical and epidemiological summaries of acute mortality rates during AMI in DM and non-DM. (**B**) Clinical and epidemiological summaries of heart dysfunction on LVEF changes post-AMI in DM and non-DM. (**C**) Summary of analyses to characterise the association of blood biomarkers in individuals with and without diabetes to the incidence and mortality of MI in the UK Biobank. (**D**) Cox proportional hazards modelling time to mortality from recruitment to the UK Biobank following MI stratified by diabetes diagnosis. (**E-F**) Effects of known MI biomarkers on time to MI mortality. Individuals stratified by diabetes diagnosis and further into the high quantile (top 30%) and bottom quantile (bottom 30%) of the biomarker levels. (**G**) Cox proportional hazards modelling time to MI incidence following MI stratified by diabetes diagnosis. (**H-I**) Effects of known MI biomarkers on time to MI incidence. Individuals stratified by diabetes diagnosis and further into the high quantile (top 30%) and bottom quantile (bottom 30%) of the biomarker levels. Cox proportional hazards regression used to determine hazard ratios and statistical significance (E-F, H-I). *P<0.05, **P<0.01, ***P<0.001, ***P<0.0001.




**B**

| Compound | | Pathway |
|---|---|---|
| higher in vG conditions | LAC | Glycolysis |
| | G3P | A central intermediate in glycolysis |
| | UMP | Nucleotide metabolism |
| | GDP | Nucleotide metabolism |
| | AMP | Nucleotide metabolism |
| | GMP | Nucleotide metabolism |

**E**

| Compound | | Pathway |
|---|---|---|
| higher in vG conditions | Sucrose | A substrate for glycolysis |
| | Benzoic Acid | Link to lipid metabolism and toxicity |
| | Beta-D-Glucosamine | Involved in sugar metabolism |
| | Lauric Acid | Link to lipid metabolism |

**Supplemental Figure 2. LC-MS and GC-MS metabolomics analysis of control, cG and vG cardiomyocytes.** (**A-B**) LC-MS analysis of the differential concentration of metabolites among control, cG and vG conditions in heatmap (**A**), with a table of higher compounds linked to metabolic pathways (**B**). (**C-E**) GC-MS analysis of the differential concentration of metabolites among control, cG and vG conditions in heatmap (**C**), the relative concentrations of the corresponding metabolite in each group under study (**D**), and a table of higher compounds linked to metabolic pathways (**E**). (n=3; 3 biological replicates each with 2 technical replicates). Statistical analysis by hierarchical clustering analysis (A, C) or partial least squares - discriminant analysis (D).