

Fig.S1. PBMC proteome of heart failure (HF) subjects (versus normal healthy (NH) controls) was deconvoluted into pathways using Ingenuity Pathway Analysis (IPA) software (Qiagen, Crawley, UK) as described in Materials and Methods, and protein spots that were differentially abundant in HF subjects (fold change > 2) were identified by mass spectrometry. Shown are **(A)** disease and disorder network of inflammation in HF subjects, including pathways such as recruitment of neutrophils, infiltration of leukocytes, chemotaxis of phagocytes, and aggregation of platelets, and **(B)** disease and bio-function network suggesting increase in ROS production and a decline in fatty acid metabolism in HF subjects. These networks were developed by Ingenuity pathway analysis of differential PBMC proteomics data (Fig. 1). In Figs.S1&S2, the intensity of red and green colors show the extent of increase and decrease respectively, in HF individuals as compared to that noted in NH controls. Gray lines indicate putative interactions or findings inconsistent with state of downstream molecule. Brown/orange node/lines and blue nodes/lines indicate activation and inhibition, respectively, of a pathway.

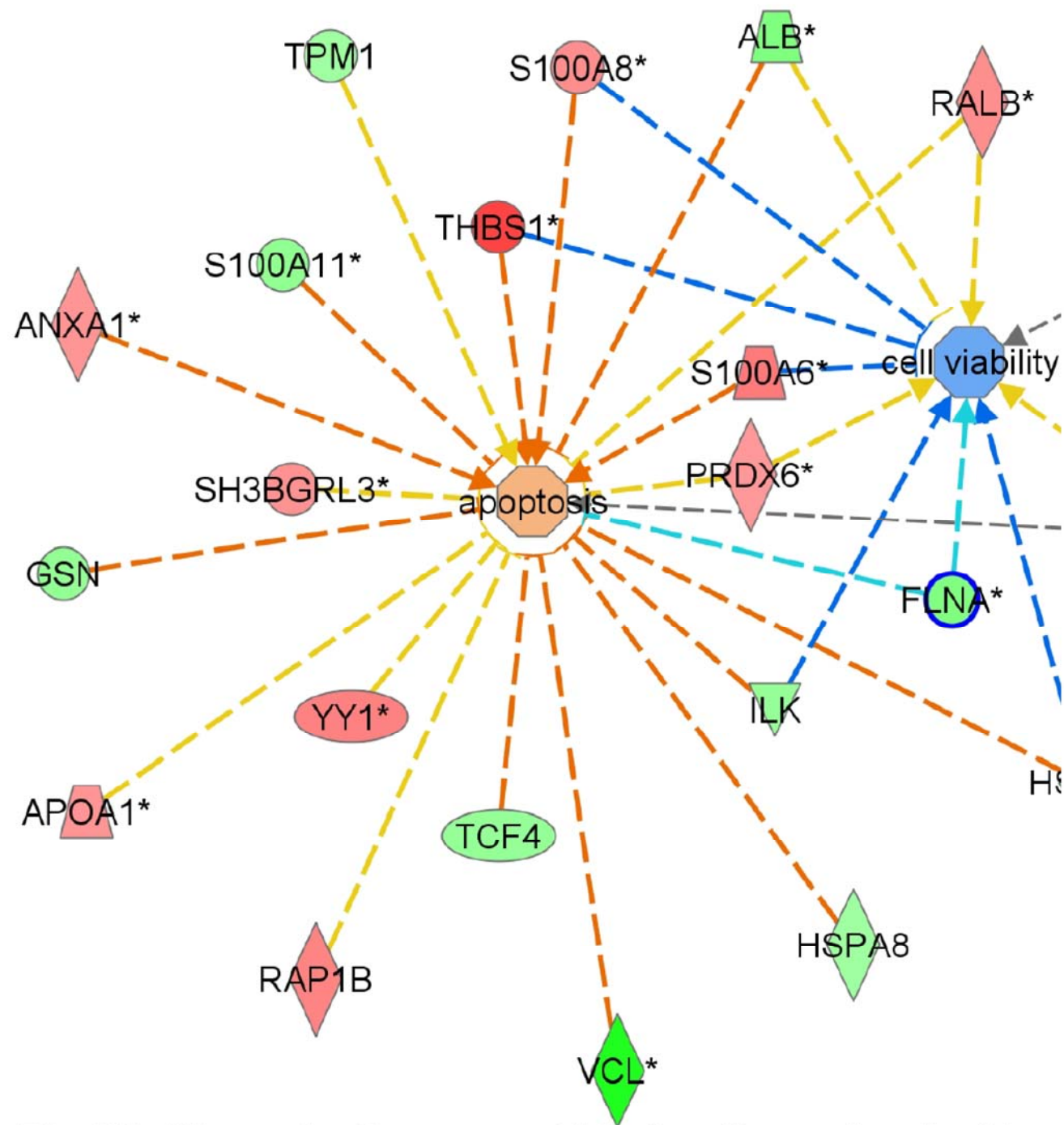


Fig.S2. Shown is disease and bio-function network of increased cell death and decreased cell survival in HF subjects versus normal healthy controls; developed by Ingenuity pathway analysis of a differential PBMC proteome abundance dataset (Table 1).

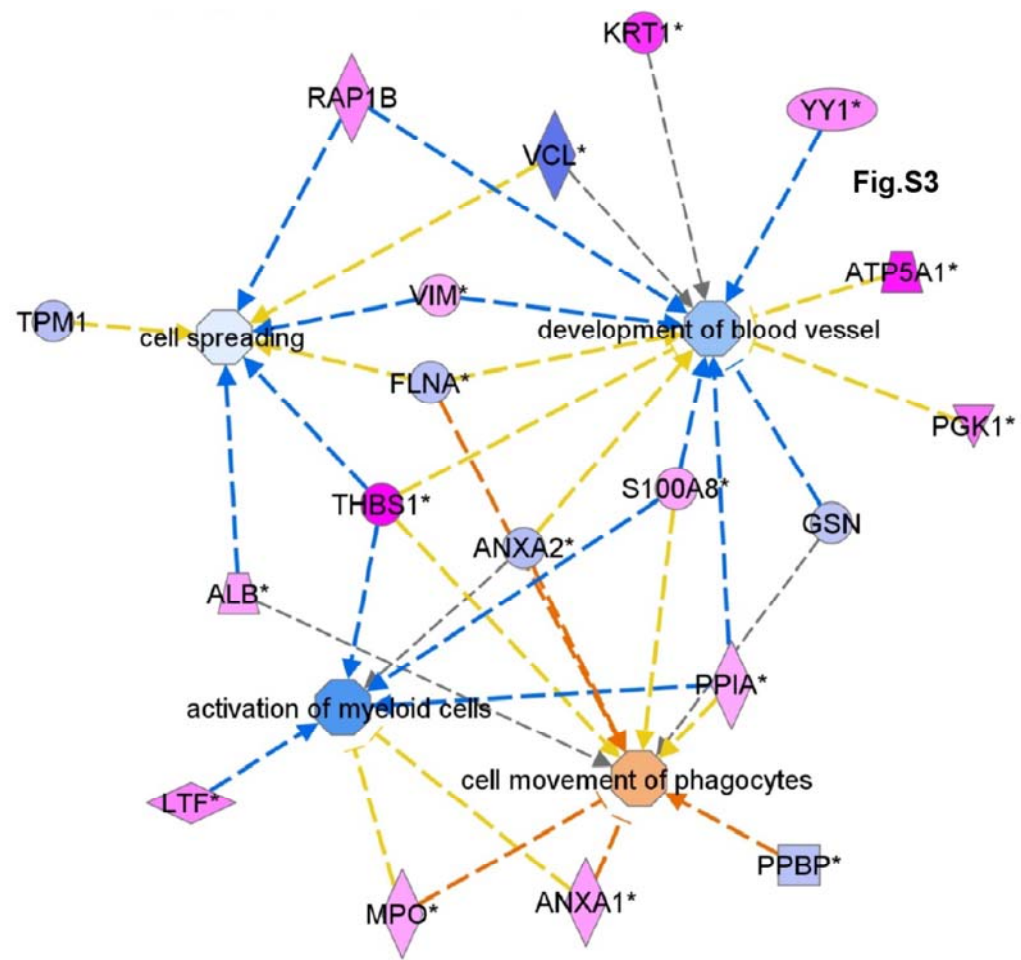


Fig.S3. PBMC proteome analysis in HF subjects (vs NH controls) described in Mateo et al. (2010). Protein spots that were not modified (change: $|\geq 1.5|$, $p < 0.05$ mass spectrometry) and disorder network analysis increased S-nitrosylation involved in migration inhibition of endothelial cells and development of blood vessel versus normal pathways were color-coded by pathway analysis: PBMC proteome analysis (Figs.S3&S4), the pink and blue colors indicate increase and decrease respectively, in compared to that of normal. Gray lines indicate predicted. Brown, blue node/lines stand for stimulation and inhibition, respectively.

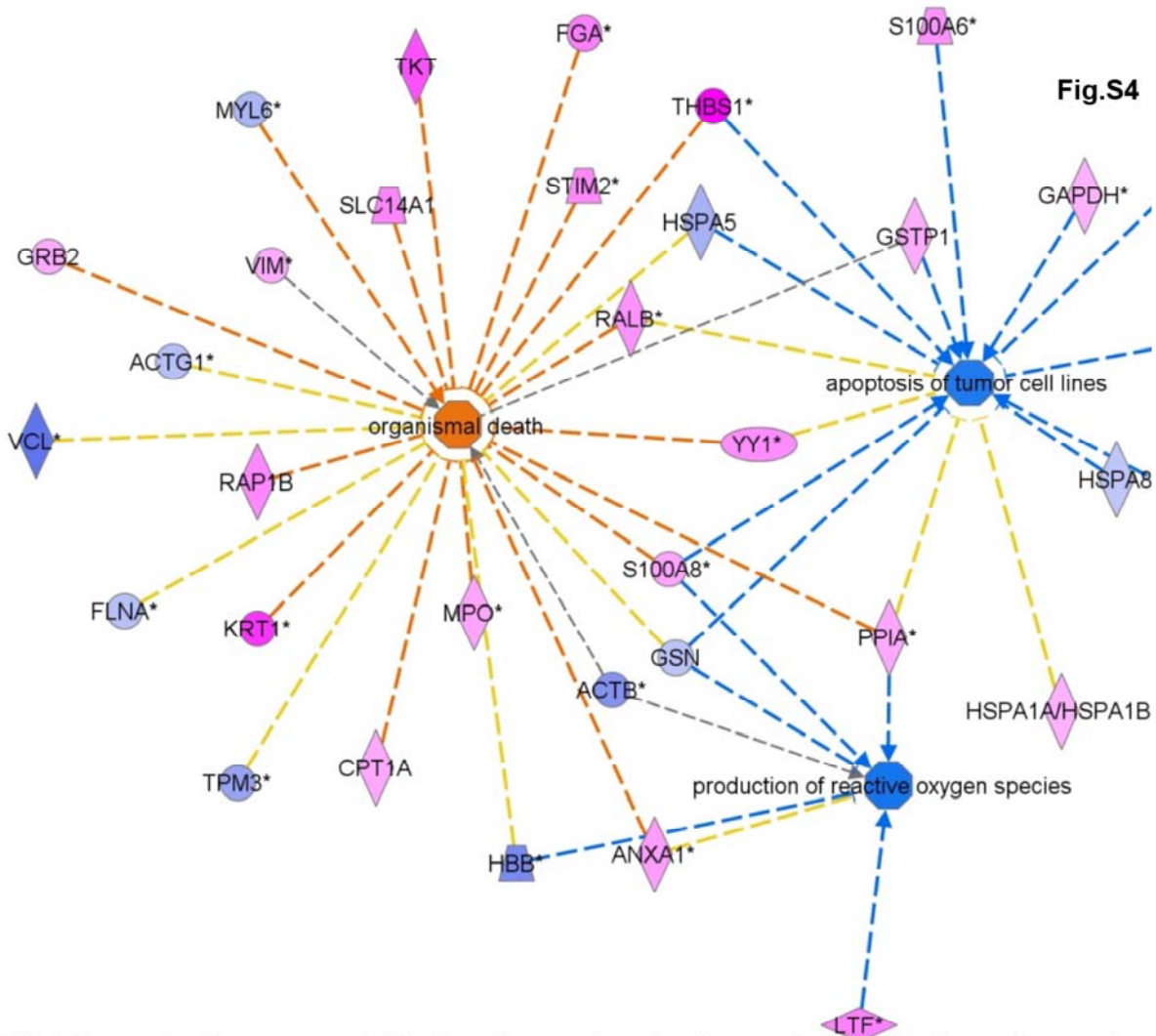


Fig.S4

Fig.S4. Shown is disease and bio-function network of organismal death, cell apoptosis of free radicals in HF subjects versus normal healthy controls; developed by In analysis of differential PBMC proteome S-NO dataset (Table 1). Note that a majority inhibit apoptosis and free radical production as well activate organismal death were suggesting that S-NO modification serves as an important mechanism in regulating ce