

SUPPLEMENTAL FIGURE LEGENDS

Fig S1. Network view of consensus clusters 4A and 4B in the module overlap graph.

Fig S2. Density plot of correlation to respective module eigengene (kME). Tissue consensus genes have significantly higher kME and are therefore more ‘hub-like’ than non-consensus genes. Mann Whitney U, $p < 2.2 \times 10^{-16}$

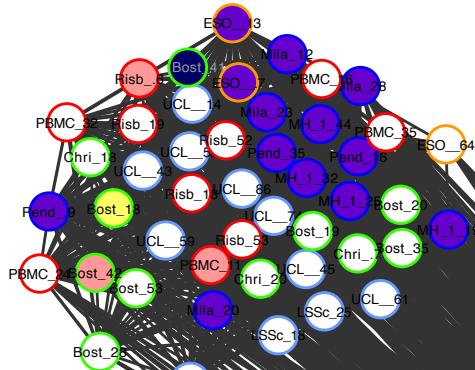
Fig S3. Fully labeled version of the lung network (Fig 3A). This file is intended to be viewed digitally.

Fig S4. Fully labeled version of the differential adjacency matrix in Fig 4B. This file is intended to be viewed digitally.

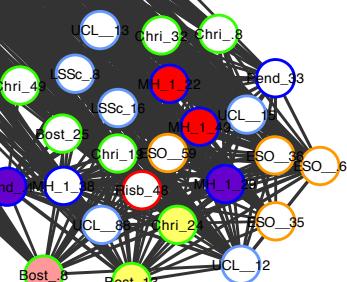
Fig S5. The differential lung network. The highly lung-specific network (minus global network and skin network) contains functional modules.

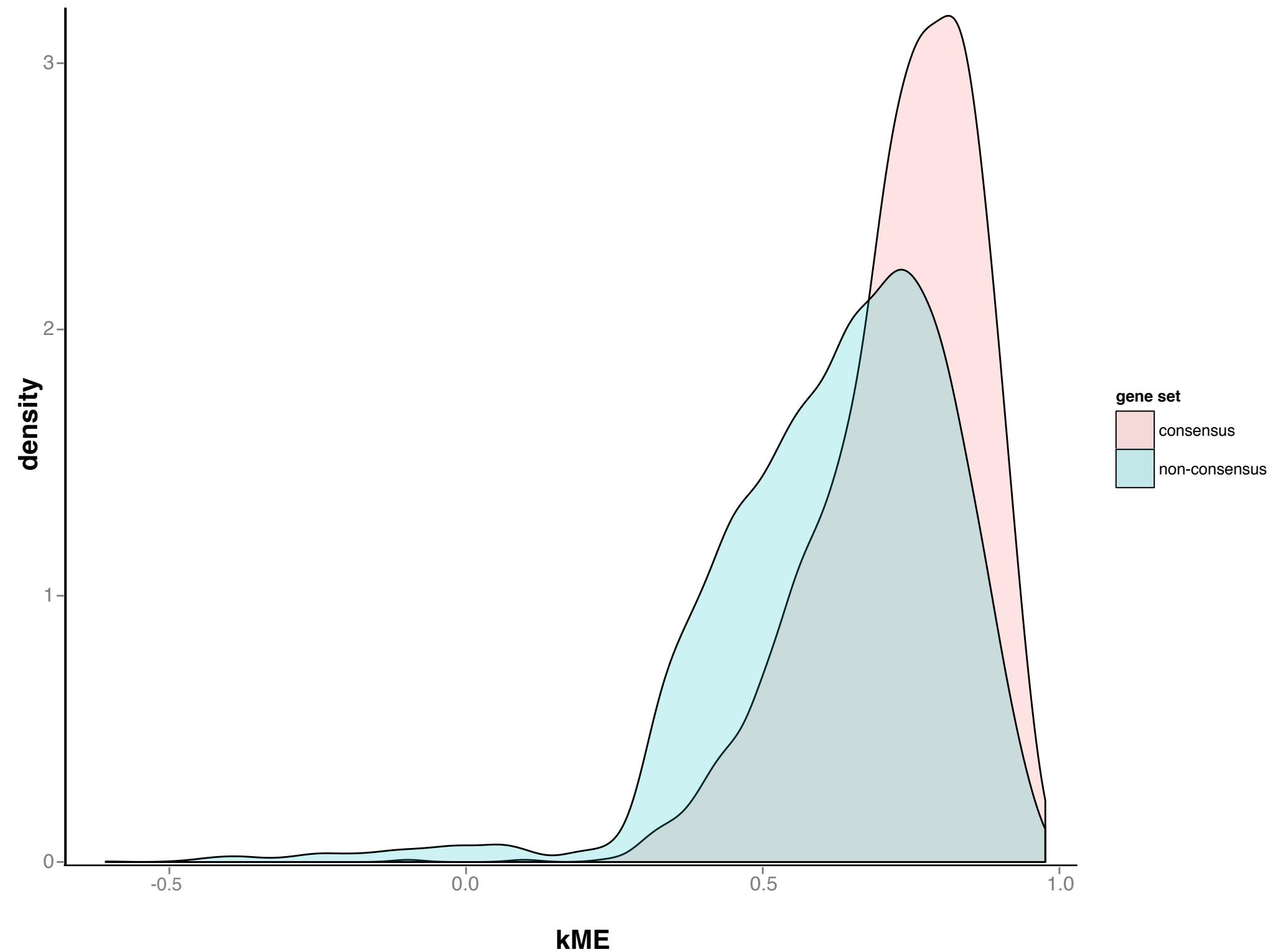
Fig S6. The lung phagolysosome/ECM disassembly functional module is more highly connected in a macrophage-specific functional genomic network than expected. Global edge weights were subtracted from the lung and macrophage networks. The black and white matrix illustrates how much weight is within a community (“on-diagonal”) or between communities (“off-diagonal”; how connected communities are to one another) by summing all edge weights in that block. We permuted the order of the adjacency matrix (edges) and assessed if the true weight within a community was more than random (red), less than random (blue), or no different than random (white). Note that there is more weight within communities in the (global subtracted) lung network than expected than random, which illustrates how the community detection algorithm utilized takes into account the expected distribution of edges/edge weights at random.

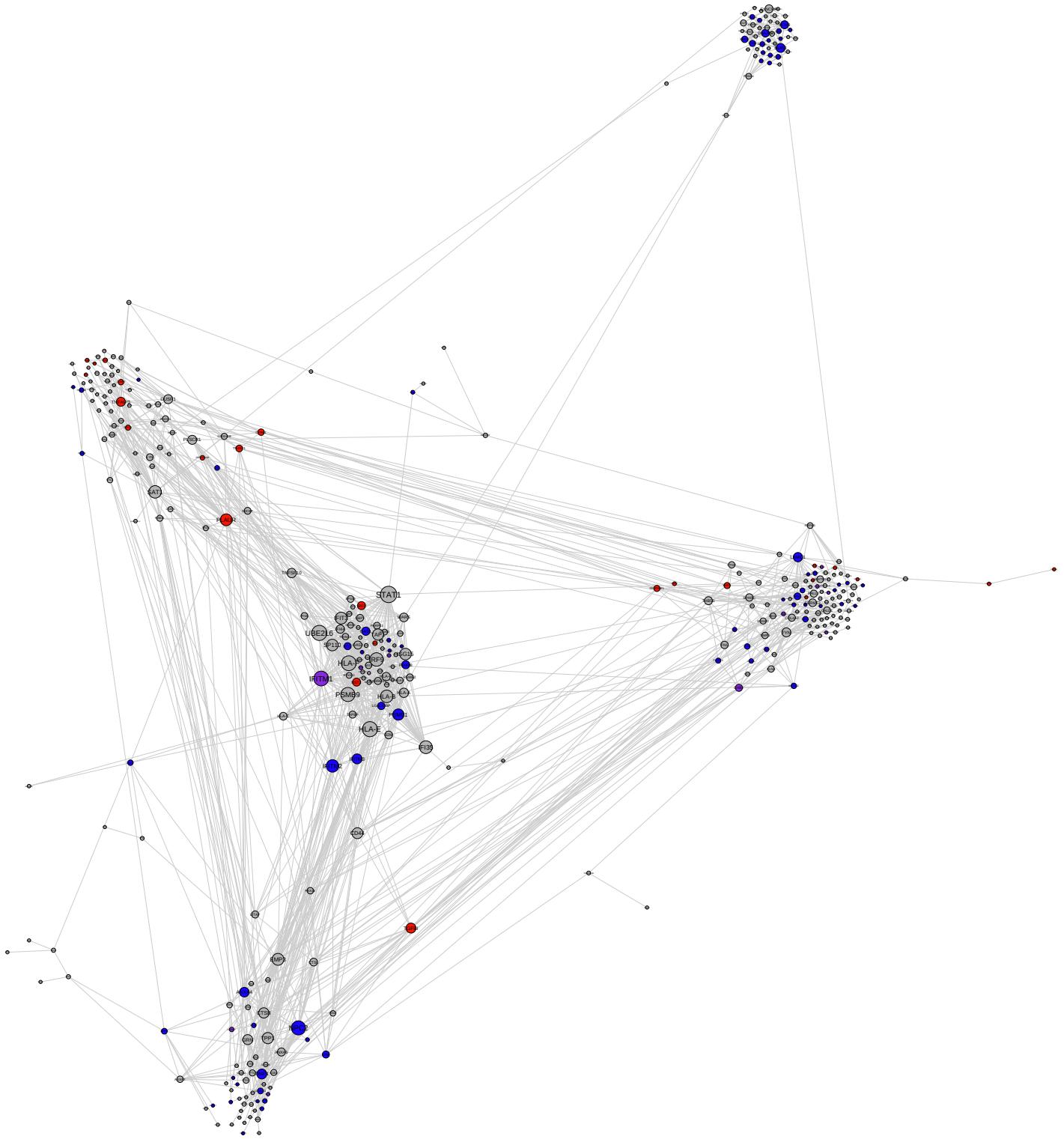
Fig S7. Analysis in Figure 5B repeated in Assassi, et al. skin dataset. Summarized expression values (mean standardized expression value) of gene sets (coexpression modules) upregulated in various MØ states. Overall, the differences between SSc skin and normal skin in this independent dataset agree with the findings in the Hinchcliff dataset, with the exception of the CL1 module; it is significantly upregulated in SSc skin in the Assassi dataset but not the Hinchcliff dataset.

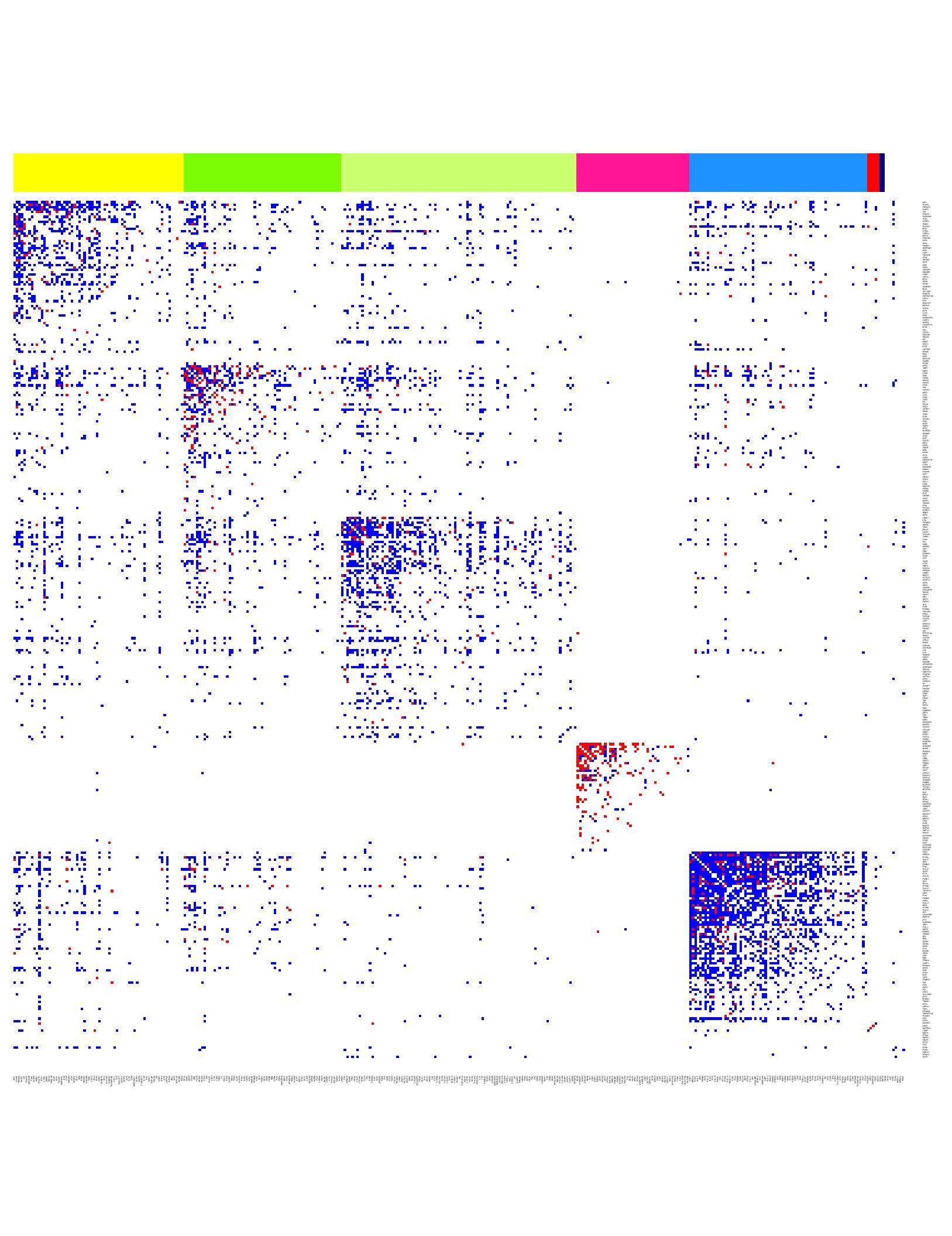
A**LEGEND**

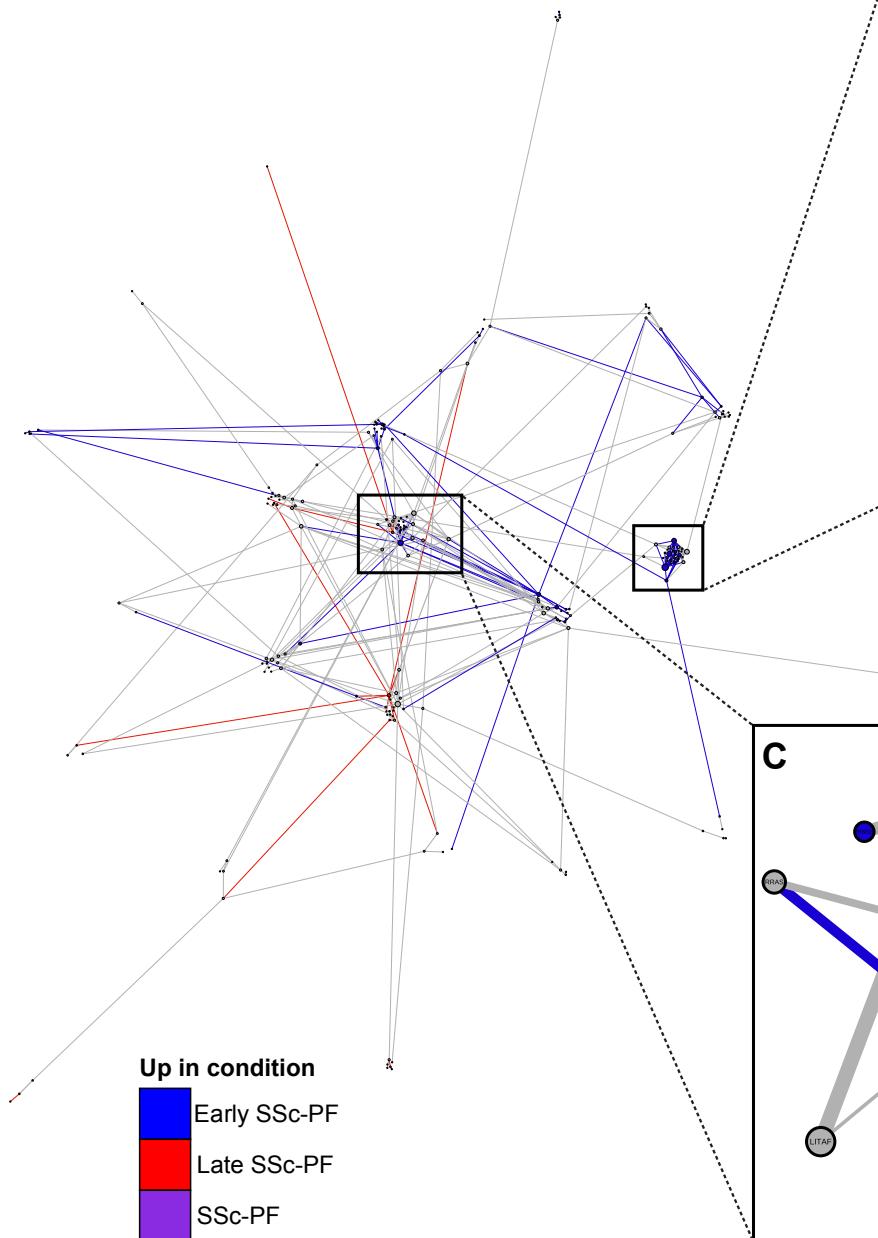
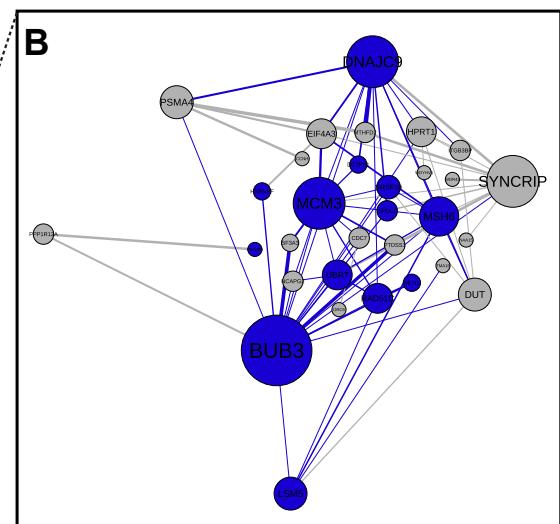
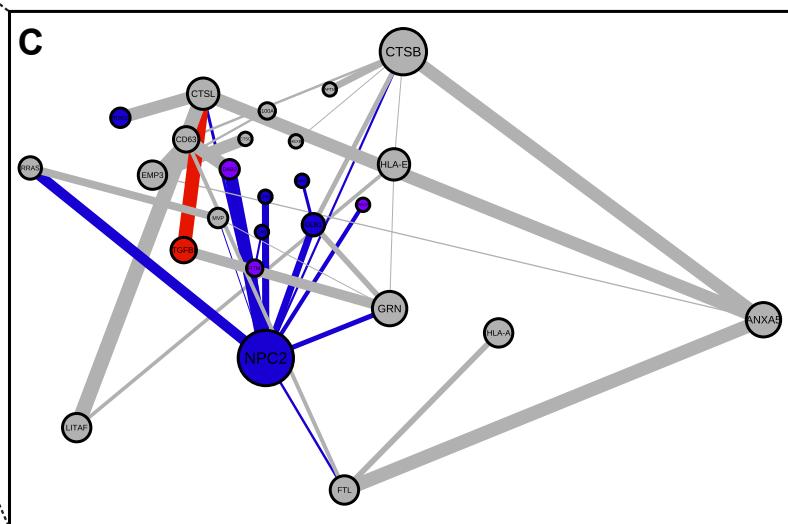
Up in inflammatory	Lung
Up in proliferative	Limited skin
Up in PAH	Skin
Up in PF	PBMC
Up in SSc	Esophagus

B

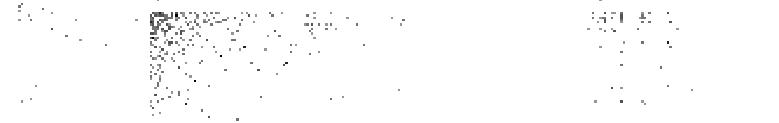




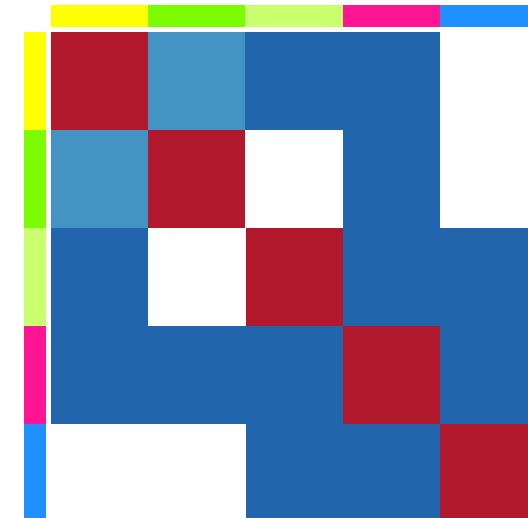
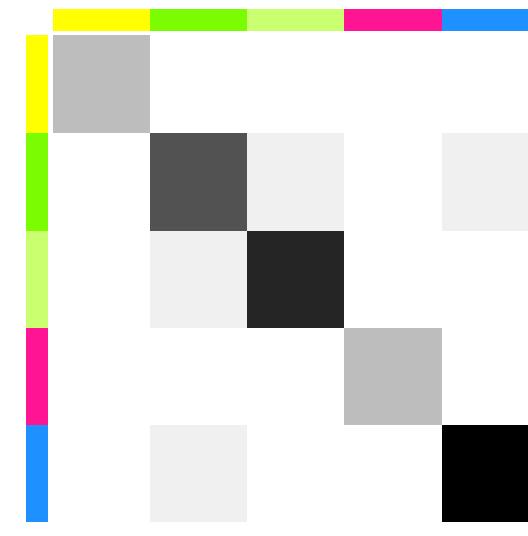


A**B****C**

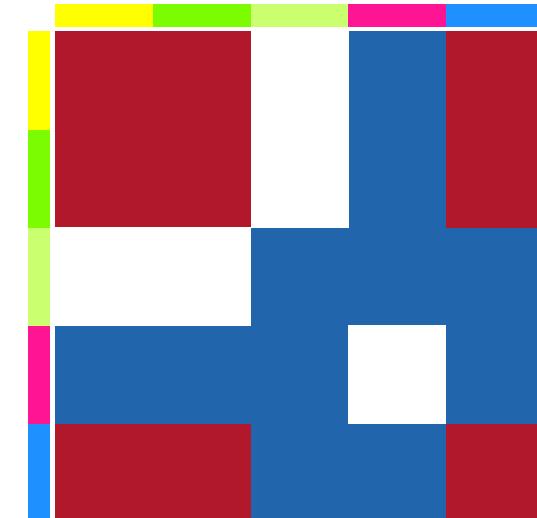
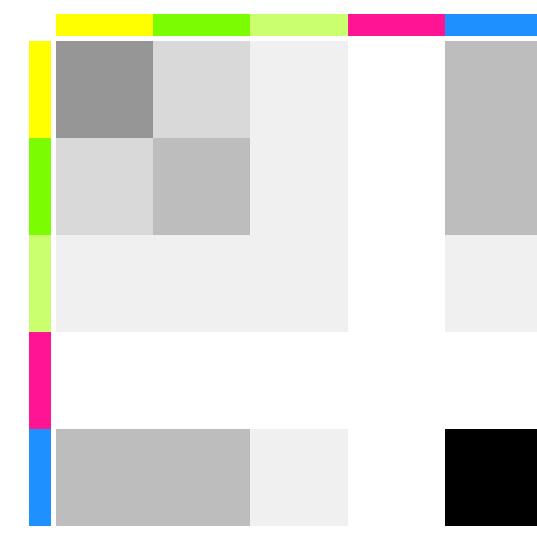
inflammatory response,
NF-kappaB signaling
phagolysosome,
ECM disassembly
fibrillar collagen;
response to TGF-beta
cell cycle
interferon;
antigen presentation



lung* network

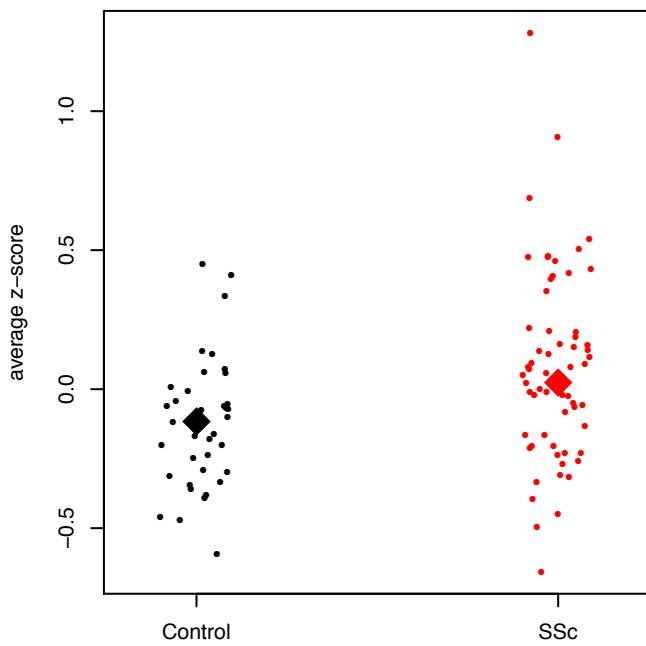


macrophage* network

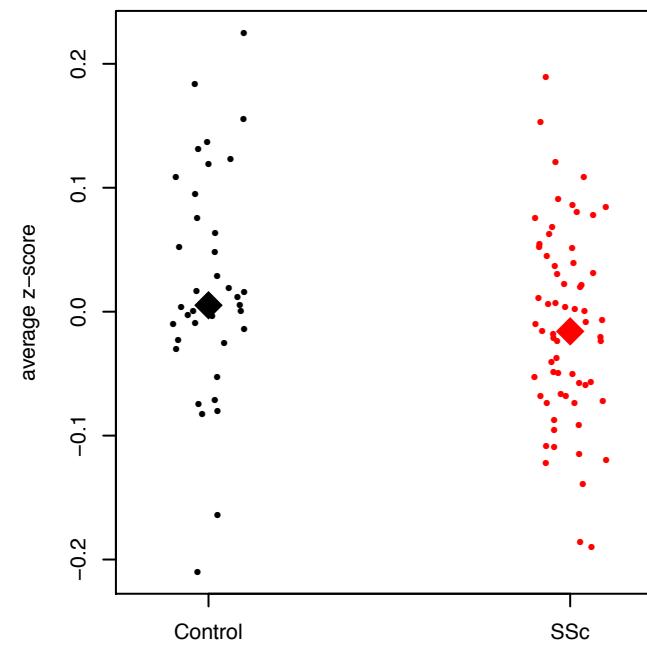


*with global subtraction

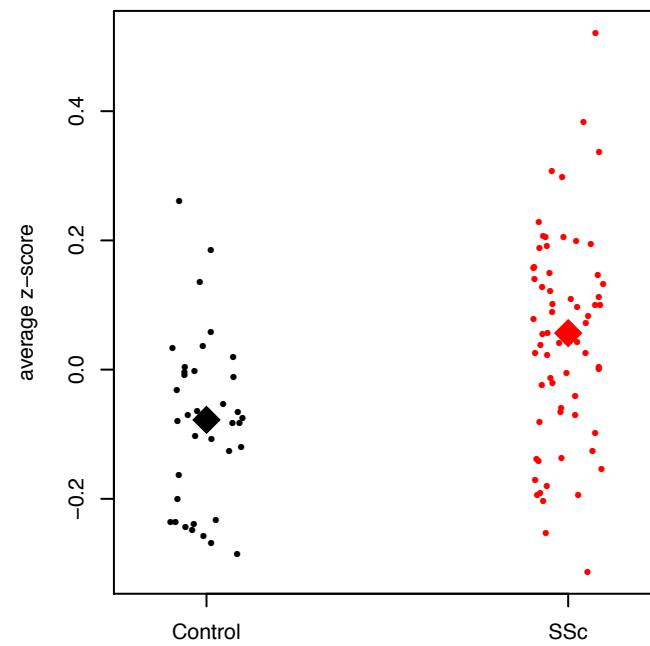
CL 1



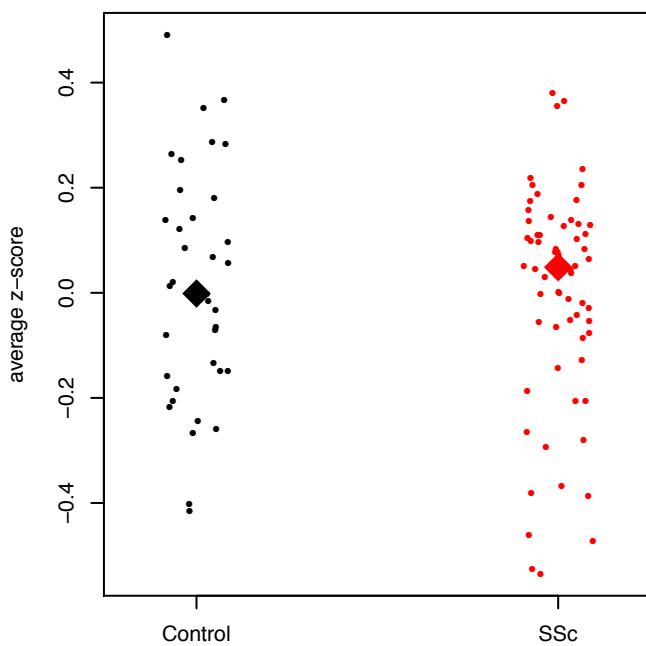
ALT 1



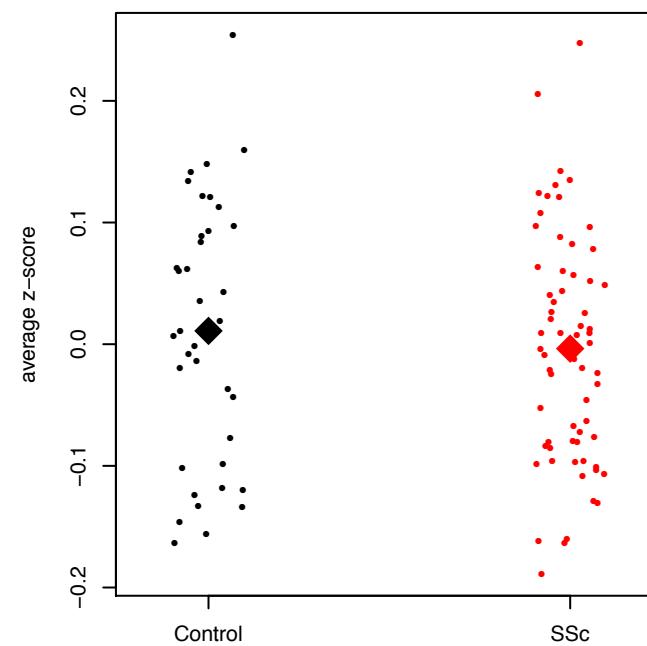
ALT 2



FFA 1



FFA 2



FFA 3

