Formation of colorectal liver metastases induces musculoskeletal and metabolic abnormalities consistent with exacerbated cachexia

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Figure S1. mC26 hosts have reduced liver glycogen. Liver glycogen ( $\mu \mathrm{g} / \mu \mathrm{l}$ ) of CD2F1 male mice (12-week old) intrasplenically injected with C26 tumor cells ( 250,000 cells/mouse in sterile PBS: mC 26 ) or an equal volume of vehicle (Sham) ( $\mathrm{n}=4-5$ ). Data are expressed as means $\pm$ SD. Significance of the differences: ${ }^{* * * *} \mathrm{p}<0.0001 \mathrm{vs}$. Sham.


Figure S2. Cardiac cachexia in C26 and mC26 tumor hosts. Heart weights normalized to initial body weight (IBW) and gene expression levels for ANP, BNP, MuRF-1, Atrogin-1 and Myh7b measured by quantitative real-time PCR and normalized to TBP levels in CD2F1 male mice (8-week old) either injected with C26 tumor cells subcutaneously ( $1,000,000$ cells/mouse in sterile PBS: C26) or intrasplenically ( 250,000 cells/mouse in sterile PBS: mC26). Control (Con for C26) and Sham (controls for mC26) were injected with an equal volume of vehicle ( $n=4-6$ ). Data are expressed as mean $\pm S D$. Significance of the differences: $\# p<0.05, \# \# p<0.01, \# \# \# p<0.001$ vs. Con; ${ }^{*} \mathrm{p}<0.05,{ }^{* *} \mathrm{p}<0.01,{ }^{* * *} \mathrm{p}<0.001$ vs. Sham.

