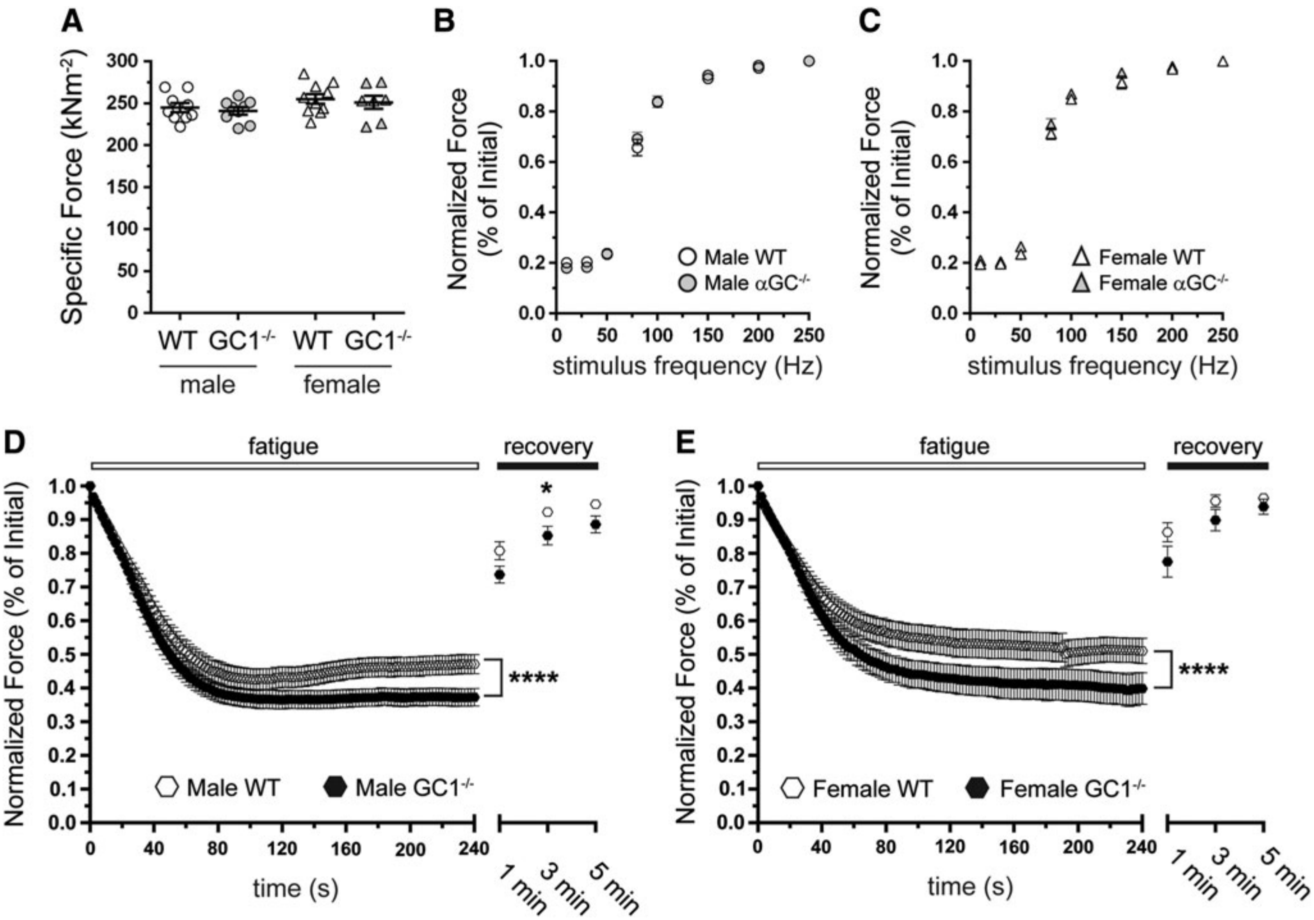

SUPPLEMENTARY FIG. S2. GC1 deficiency promotes skeletal muscle fatigue in male and female mice. (A) Specific force output in TA muscles from male and female WT and $GC1^{-/-}$ mice. Specific force was similar between WT and $GC1^{-/-}$ male and female TA muscles, indicating that GC1 is dispensable for normal muscle strength. $n = 9$ for male groups, $n = 10$ and 7 for female WT and $GC1^{-/-}$ groups, respectively. **(B)** Force–frequency curves for TA muscles from male WT and $GC1^{-/-}$ mice. Force output at different frequencies of stimulation was unaffected by loss of active GC1 consistent with normal gross neuromuscular synapse function. $n = 11$ for both groups. **(C)** Force–frequency curves for TA muscles from female WT and $GC1^{-/-}$ mice. As seen in males, force–frequency curves were similar between WT and $GC1^{-/-}$ mice, suggesting that GC1 is dispensable for neuromuscular transmission. $n = 7$ for both groups. **(D)** Contraction-induced fatigue resistance of TA muscles from male WT and $GC1^{-/-}$ mice. GC1-deficient TA muscles were unable to sustain normal output during repeated stimulation and showed impaired force recovery. $n = 19$ and 21 for WT and $GC1^{-/-}$ groups, respectively. **(E)** Contraction-induced fatigue resistance of TA muscles from female WT and $GC1^{-/-}$ mice. As seen with males, GC1-deficient female TA muscles exhibited force deficits during repeated stimulation; however, force recovery was not significantly impacted, suggesting sex-specific roles for GC1 in force recovery after exercise. $n = 20$ and 13 for WT and $GC1^{-/-}$ groups, respectively. **(D, E)** $^{*}p < 0.05$; $^{****}p < 0.0001$ from regular two-way ANOVA using genotype and time as variables with Tukey's multiple comparison test.