

FIG S1 Relative *acuM* and *acuK* mRNA levels in mutants lacking *acuK* and/or *acuM*. The indicated *A. fumigatus* strains were grown in RPMI 1640 broth for 24 h, after which total RNA was isolated. The transcript abundance of *acuM* and *acuK* was measured using real-time RT-PCR and normalized to *TEF1* mRNA levels. Results are the mean ± SD of 3 biological replicates, each tested in triplicate.

WFL-FITC staining

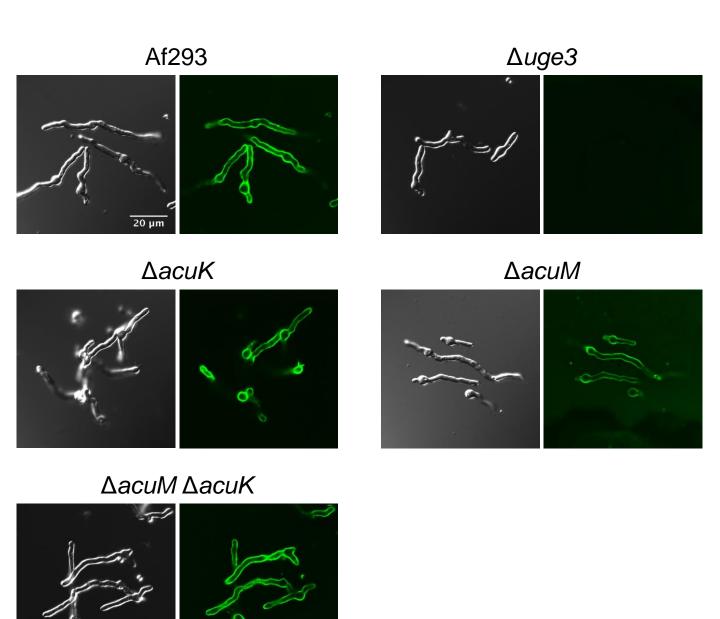


FIG S2 Deletion of *acuK* and *acuM* has no effect of the surface expression of galactosaminogalactan (GAG). The indicated strains were germinated for 9 h and then stained with FITC-labeled *Wisteria fluoribunda* lectin (WFL) to detect surface-exposed GAG. The strains were then imaged by differential interference contrast (left) and confocal microscopy (right). The GAG-deficient Δ*uge3* strain was used as a negative control.

Fig. S3

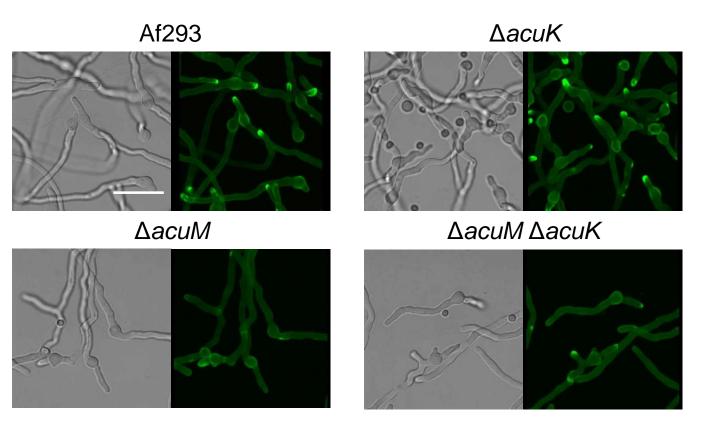
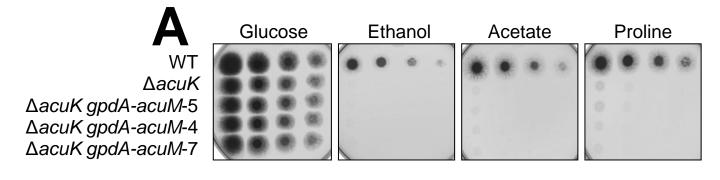


FIG S3 Deletion of acuK and acuM has no effect on cell wall β -glucan. The indicated strains were germinated for 9 h and then stained aniline blue to detect β -glucan. The strains were then imaged by differential interference contrast (left) and confocal microscopy (right).

Fig. S4



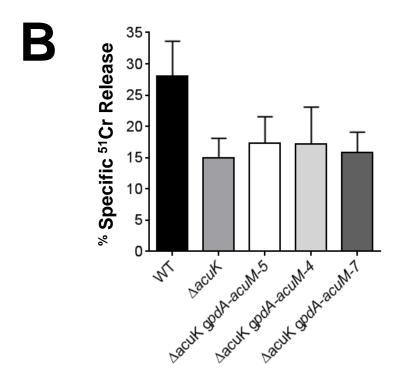


FIG S4 Effects of overexpression of *acuM* on the growth of the $\Delta acuK$ mutant on gluconeogenic carbon sources (A) and damage to a pulmonary epithelial cell line. (A) Serial 10-fold dilution of the indicated strains were plated on agar containing glucose, ethanol, acetate, or proline as the carbon source. The plates were imaged after incubation at 37°C for 48 h. (B) The A549 pulmonary epithelial cell line was infected with the indicated strains of *A. fumigatus* for 20 h, after which the extent epithelial cell damage was assessed by a 51 Cr release assay. Results are the mean \pm SD of 3 independent experiments, each performed in triplicate.