Glucolipids in *Bacillus subtilis* are synthesized by UgtP processively transferring glucose from UDP-glucose to diacylglycerol. Here we conclude that the abnormal morphology of a *ugtP* mutant is caused by lack of glucolipids, since the same morphology arises after abolition of glucolipid production by disruption of *pgcA* and *gtaB*, which are involved in UDP-glucose synthesis. Conversely, expression of a monoglucosyldiacylglycerol (MGlcDG) produced by 1,2-diacylglycerol 3-glucosyltransferase from *Acholeplasma laidlawii* (alMGS) almost completely suppressed the *ugtP* disruptant phenotype. Activation of extracytoplasmic function (ECF) sigmas (SigM, SigV, and SigX) in the *ugtP* mutant was decreased by alMGS expression, and was suppressed to low levels by $MgSO_4$ addition. When alMGS and alDGS (*A. laidlawii* 1,2-diacylglycerol-3-glucose (1-2)-glucosyltransferase producing diglucosyldiacylglycerol (DGlcDG)) were simultaneously expressed, SigX activation was repressed to wild type level. These observations suggest that MGlcDG molecules are required for maintenance of *B. subtilis* cell shape and regulation of ECF sigmas, and DGlcDG regulates SigX activity.

The abnormal morphology of the *B. subtilis ugtP* mutant was suppressed by expression of heterologous MGlcDG synthase (left). Effect of heterologous glucolipids on activitiy of ECF sigmas (right).