Figure S1  Network diagram of diguanylate cyclase (DGC)-encoding pathways underpinning the evolution of the WS phenotype and their regulation (adapted from Figure 2, McDonald et al. 2009). Overproduction of c-di-GMP results in overproduction of cellulose (via activation of enzymes encoded by wss genes) and other adhesive factors that form the structural matrix of the WS biofilm (Spiers et al. 2002; Goymer et al. 2006). The ancestral SBW25 genome contains 39 putative DGCs (Silby et al. 2009), each in principle capable of synthesizing the production of c-di-GMP, and yet WS genotypes arise most commonly as a consequence of mutations in just three DGC-containing pathways: Wsp, Aws, and Mws (McDonald et al. 2009). In each instance, the causal mutations are most commonly in the negative regulatory component: wspF, awsX, and the phosphodiesterase domain of mwsR. WS mutant strains used in this study (see Table S1) are indicated in red.