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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.