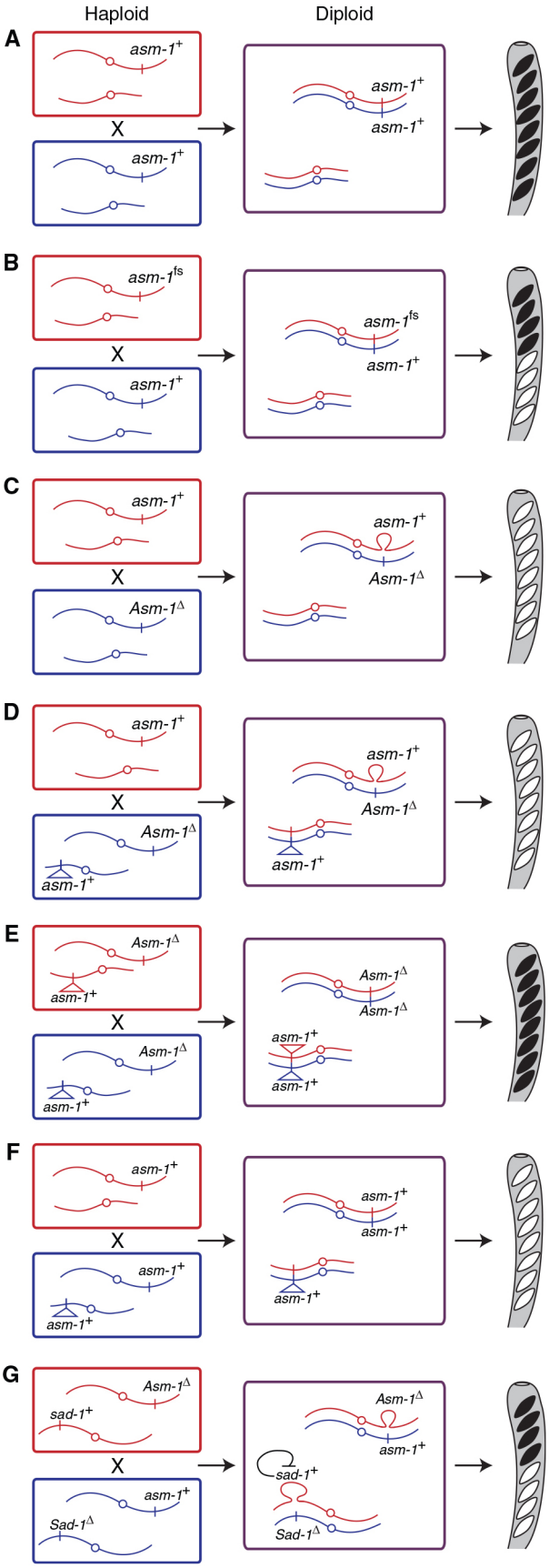

Figure 8. Discovery and characterization of meiotic silencing. Key genetic experiments are illustrated using the *Ascospore maturation-1* (*Asm-1*) gene, as a reporter. For each cross, the relevant genotype of the haploid parents of *mating type A* (red boxes) or *mating type a* (blue boxes) is shown on the *left*, and cartoons showing the predicted chromosome pairing in the diploid cell (violet boxes) is shown on the *right*. The phenotypes of resulting asci are presented on the *far right*. Black represents mature (typically viable) ascospores and white represents immature (inviable) ascospores. (*A*) Wild-type cross. (*B*) 4:4 segregation of ascospores from a heterozygous cross of wild type and a frameshift mutant in which alleles can pair and no meiotic silencing occurs. (*C*) Cross of strains with wild-type and deletion alleles triggers meiotic silencing. (*D*) Meiotic silencing is not rescued by ectopic wild-type allele, indicating that the developmental defect is not due to haploinsufficiency. (*E*) Allelic (pairable) ectopic copies *asm-1*$^{+}$ in crossing partners rescue *Asm-1*$^{-}$ defect. (*F*) Presence of an unpaired allele triggers silencing of all *asm-1*$^{+}$ alleles (paired and unpaired) in meiosis. (*G*) Silencing of the suppressor of ascospore dominance (*Sad-1*), because of a *Sad-1* deletion in one parent, suppresses meiotic silencing.