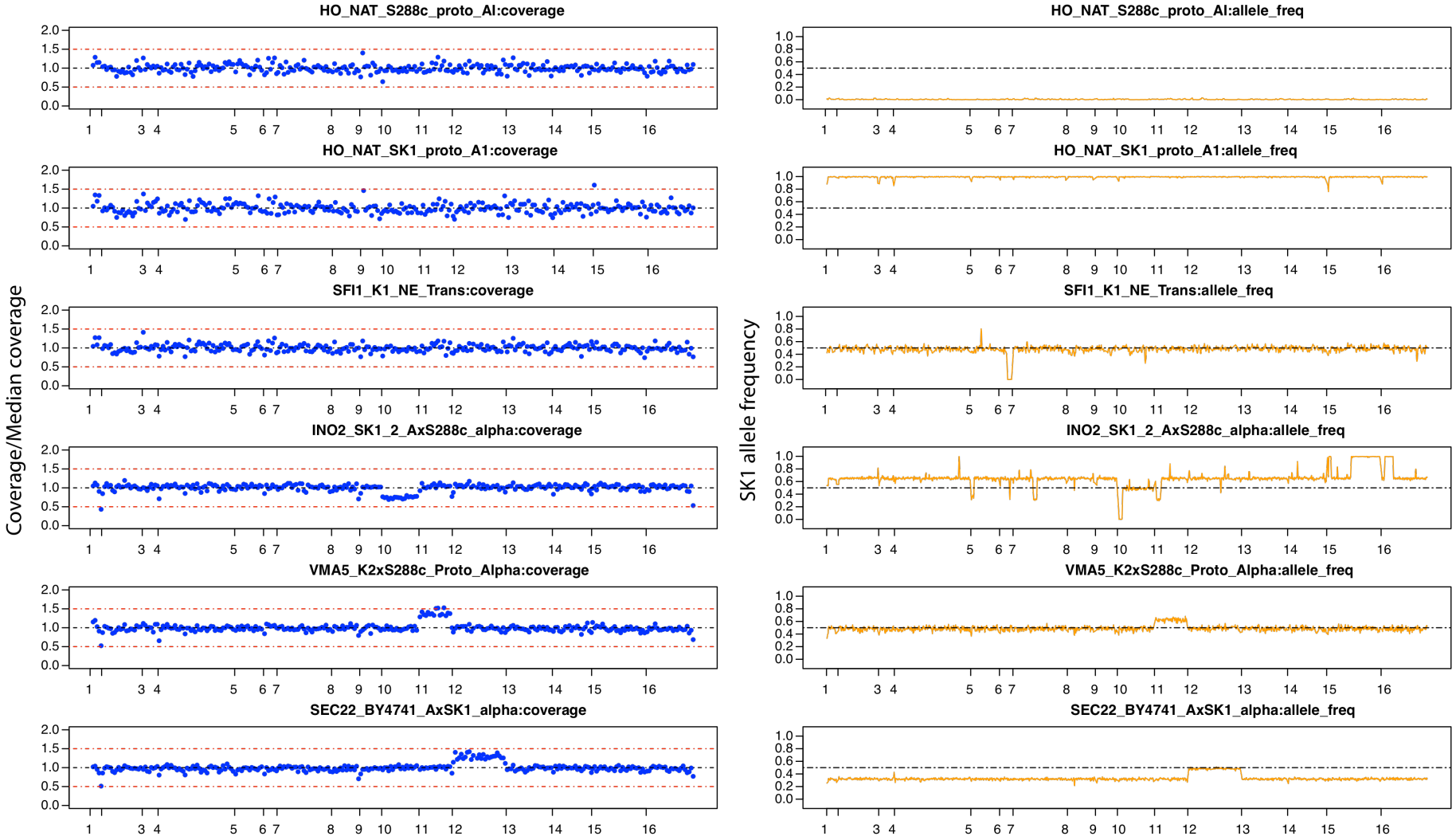
HO_NAT_S288c_proto_AI:coverage
HO_NAT_SK1_proto_A1:coverage
SFI1_K1_NE_Trans:coverage
INO2_SK1_2_AxS288c_alpha:coverage
VMA5_K2xS288c_Proto_Alpha:coverage
SEC22_BY4741_AxSK1_alpha:coverage
HO_NAT_S288c_proto_AI:allele_freq
HO_NAT_SK1_proto_A1:allele_freq
SFI1_K1_NE_Trans:allele_freq
INO2_SK1_2_AxS288c_alpha:allele_freq
VMA5_K2xS288c_Proto_Alpha:allele_freq
SEC22_BY4741_AxSK1_alpha:allele_freq
Coverage/Median coverage
SK1 allele frequency

Figure S9: Resequencing of 50 RHS strains reveals a high rate of aneuploidy.

Except for cantharidin resistance, RHS results did not identify the QTGs found by ISA or BSA. By resequencing 50 of the original RHS deletion strains we found that 17 carried chromosomal aberrations (see examples above and Table S5 for summary). Left panels shows copy number variation and right panel shows allele frequency (100% SK1 = 1), for each sample along the different chromosomes. HO_NAT_S288c_protoAI and HO_NAT_SK1_protoA1 are parental strains used in the construction of RHS strains and showed no abnormalities. The remaining four strains showed either a loss of heterozygosity (SF1_K1_NE_Trans), a copy number variation (VMA5_K2xS288c_Proto_Alpha, chr 11, 3 copies) or a combination of both (INO2_SK1_2_AxS288c_alpha, a triploid across all chromosomes except for chr 10 and loss of heterozygosity on chr 15). Among the sequenced strains, seven triploid strains could be detected, likely originating from mating of a diploid with a haploid strain (e.g. two genome copies of S96, one of SK1). In addition, four out of 12 randomly picked strains harbored an extra chromosome, suggesting a widespread aneuploidy in the RHS collection.