Table S3. Suppression of high temperature arrest (HTA) by genetic mutations in *mes-4* and *mrg-1*

Parent genotype	Percentage of L1 progeny that grew to adulthood at 26°C (*n*)
lin-15B(n744)	0 (43)
mes-4(bn85)	85 (34)
mes-4(bn85)/DnT1GFP; lin-15B(n744)	0 (127)
mes-4(bn85); lin-15B(n744)	35 (107)
mrg-1(qa6200)/qC1GFP	23 (232)
mrg-1(qa6200)/qC1GFP; lin-15B(n744)	23 (535)

mes-4(bn85) was able to suppress the HTA phenotype, but only did so in a maternal effect manner. *mes-4(bn85); lin-15B(n744)* homozygous F1 progeny from a *mes-4(bn85)/+* heterozygous mother showed no suppression of HTA, whereas *mes-4(bn85); lin-15B(n744)* homozygous F1 progeny from a *mes-4(bn85); lin-15B(n744)* homozygous mothers showed some suppression of the HTA phenotype (lines 1-4). By contrast, *mrg-1(qa6200)* was able to suppress *lin-15B(n744)* HTA in a non-maternal effect manner such that an equivalent amount of *mrg-1(qa6200)* homozygous F1 progeny grew to adulthood from *mrg-1(qa6200)/qC1GFP* and *mrg-1(qa6200)/qC1GFP; lin-15B(n744)* mothers (lines 5,6).