Supporting Information

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Fig. 51. Generation of $dtrpA1^{fs}$ mutant and wild-type control ($dtrpA1^{wtR}$) strains. $dtrpA1^{ins}$ was generated via homologous recombination-mediated gene targeting (48). $dtrpA1^{ins}$ tandem duplication was subsequently reduced to a single copy through I-Cre-I break–induced recombination (29). Recombination upstream of frameshift mutation generated $dtrpA1^{fs}$, whereas recombination downstream of frameshift mutation regenerated a wild-type dtrpA1 allele, $dtrpA1^{wtR}$. The $dtrpA1^{wtR}$ chromosome should be identical to $dtrpA1^{ins}$ and $dtrpA1^{fs}$ except for sequences within dtrpA1. $dtrpA1^{wtR}$ serves as a genetic control analogous to the precise excision of a transposon. Letter X denotes approximate crossover locations. Asterisk indicates frameshift mutation in exon 4. See Materials and Methods for details.



Fig. 52. (*A*) Wild-type (n = 7) and $dtrpA1^{ins}$ (n = 7) robustly avoid *n*-octyl acetate. *, P < 0.05, unpaired *t* test. (*B*) Wild-type (n = 6) and $dtrpA1^{ins}$ (n = 6) are equally attracted to propionic acid. (*C*) Wild-type (n = 10) and $dtrpA1^{ins}$ (n = 10) exhibit indistinguishable phototactic behavior. (*D*) Warm avoidance of wild-type (n = 10), original $dtrpA1^{ins}$ (n = 35) and backcrossed $dtrpA1^{ins}$ strains. **, P < 0.01, Tukey-Kramer HSD.