LETTER

# Longevity of *Indy* mutant *Drosophila* not attributable to *Indy* mutation

In their recent study in PNAS, Neretti et al. (1) identify mitochondrial defects in male *Drosophila melanogaster* that are heterozygous for the mutation *Indy*206. This allele lowers the expression of *Indy*, which encodes a Krebs cycle intermediate transporter and was previously proposed to increase adult fly lifespan (2). The authors propose an interesting model in which mitochondria work at lower rate but are present in greater density. This model results in unchanged ATP levels but less ROS production, potentially accounting for the longevity of this strain.

Although this model may be correct, the role of the *Indy* gene in these effects on lifespan is highly doubtful. Although males of the mutant strain Neretti et al. used (*Indy*206 in the Canton-S genetic background) are verifiably long-lived (2,3), we previously demonstrated that the longevity of this strain does not segregate with the *Indy* mutation (3). Instead, it largely depends on the presence of a tetracycline-dependent agent (probably *Wolbachia*), plus some other X-chromosomal locus (or loci) (3). Regrettably, it seems that Neretti et al. have attempted to brush these inconvenient facts under the rug, and we feel that we should draw attention to this. Clearly, if mutation of *Indy* does not slow aging, then the mitochondrial defects in the *Indy* strain are either not caused by *Indy* or, if they are, they do not cause the longevity.

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2. Rogina B, et al. (2000) Extended life-span conferred by cotransporter gene mutations in *Drosophila*. *Science* 290:2137-2140.
3. Toivonen JM, et al. (2007) No influence of *Indy* on lifespan in *Drosophila* after correction for genetic and cytoplasmic background effects. *PLoS Genet* 3:e95.

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