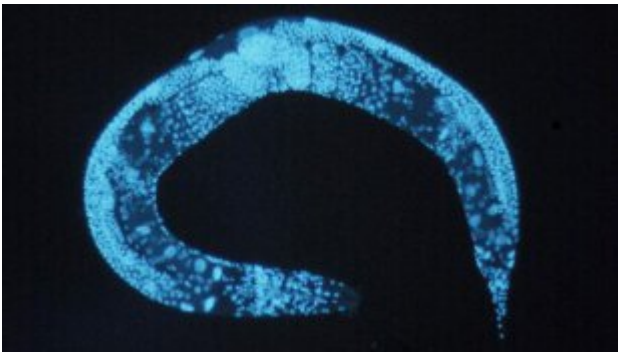

[News & Opinion](#)

The Tradeoff of Stress

For nematode worms, a bigger stress response means a healthier, longer life, but fewer babies.

By Ruth Williams | December 15, 2011



C. elegans Wikimedia Commons, National Human Genome Research Institute

Success often comes with its fair share of stress, and it's no different for the model nematode worm, *Caenorhabditis elegans*. A report published today (December 15) in [Science Express](#) reveals that worms that have a greater stress response are better at coping with deleterious mutations than their weakly-responding counterparts. But at a cost—these healthier, stress-responding worms are not as good at reproducing.

“We know that tradeoffs are important in evolution,” said [Joanna Masek](#) of the University of Arizona, who was not involved in the study, “but what this paper shows is that not only is there a tradeoff between stress response and having lots of babies, but that tradeoff can come down to a difference in just one, or a handful of factors.”

The factors in question are chaperones—proteins that assist in the folding, assembly, and stabilization of other cellular proteins and molecules. When a stressor, such as heat, knocks cellular proteins out-of-kilter, chaperones get busy folding new ones. In essence, chaperones are buffers of environmental stress, said Ben Lehner of the Centre for Genomic Regulation in Barcelona, Spain, who led the study.

In the current study, Lehner and his colleagues found that chaperones don't just buffer against stress, but also the effects of mutations. The team studied worms carrying mutations in particular developmental genes that caused a variety of phenotypes from embryonic or larval death to defects in vulva development, gonad migration, and body morphology. But not all of the worms were so bad off; those exposed to a brief period of high heat tended to display milder phenotypes—such as greater chance of survival, or improved anatomical measures.

Interestingly, not all worms benefited from the stress equally. Measuring the animals' chaperone levels, the researchers found that those that produced greater numbers of the protective proteins were better protected from the negative consequences of the mutation. This variation in chaperone levels occurred despite the worms being genetically identical and being raised in the same environment.

In addition to the effects of genes and environment on an organism's phenotype, “there is another

factor, a third one, that we may have ignored a little bit—and that is sheer luck,” said [Kevin Verstrepen](#) of the Katholieke Universiteit in Leuven, Belgium, who did not participate in the study. “The question is, why on earth don’t all animals have high chaperone levels all the time. It seems like a good thing.” Indeed, besides reduced effects of the mutation identified in this study, animals with high-chaperone levels have been shown to live longer.

But it wasn’t all good news for the worms with high chaperone levels. They also developed more slowly, thus reaching reproductive maturity at a later age. “So under low stress conditions they would be out-competed by the ones with low chaperone levels,” Lehner said.

The results of the study could have implications for humans, Verstrepen suggested. “Humans are not worms and we cannot experiment with them as we do worms, but that said, all these things that are happening in worms—the same proteins and stress responses—can also occur in humans,” he said, “so I don’t see any reason why this would not happen in humans.”

Lehner was less willing to speculate on the relevance of the link between stress response and reproduction, but suggested the finding that genetically and environmentally identical individuals can still vary in gene expression is certainly relevant. “What it tells us is that even if you understand the genetics of a disease completely, you may never be able to predict for any individual what will actually happen, unless you start measuring inter-individual variation in gene expression and [looking at] previous life history,” he said.

M.O. Casanueva et al., “Fitness trade-offs and environmentally induced mutation buffering in isogenic *C. elegans*,” [Science Express](#), 10.1126/science.1213491, 2011.

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