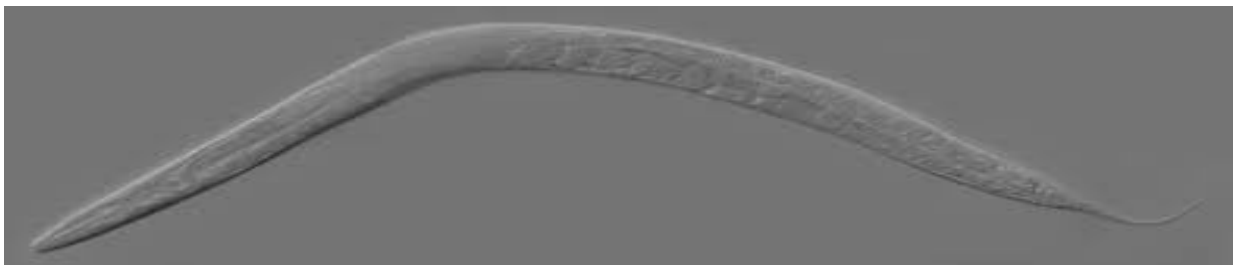
# DBTINII Study finds why dietary restriction fails in cep-1(gk138) mutant of *C. elegans* p53

New Delhi, Nov 19: Dietary Restriction (DR) is known to increase life span and impart health benefits to a wide range of metazoans, including the powerful genetic model system Caenorhabditis elegans. As a result, *C. elegans* has been used extensively to understand the mechanisms of DR. DR can be implemented in C. elegans either using genetic means or by non-genetic interventions like physically restricting diet. The p53 protein is a well-known tumour suppressor that has an important role in maintaining genome integrity by inducing DNA repair, cell cycle arrest and apoptosis in response to genotoxic stress. Various model organisms like mice, flies and worms have been exploited to investigate the role of p53 in the aging process.



In a new study, researchers at DBT-National Institute of Immunology (DBT-NII) sought to look at the role of the worm ortholog of p53, cep-1in DR-mediated lifespan extension. Previous studies have reported that knocking down cep-1 by mutation or RNAi leads to a small but significant increase in lifespan, in a context dependent manner. Most of these studies have been performed using cep-1(gk138) allele. The TJ1 strain that was used in these studies was prepared by backcrossing cep-1(gk138) ten times with wild-type N2. Here, they show that in the TJ1 stain, the genetic modes of DR do not work as the process of autophagy and the expression of cytoprotective xenobiotic detoxification genes fail to get upregulated. However, on further backcrossing TJ1, they found that the life span effects were lost, suggesting that background mutations may be the reason for the earlier suppression seen in our experiments. they validated the results in other cep-1 alleles and additionally, defined a bona fide phenotype controlled by cep-1. Considering the fact that TJ1 strain has been extensively used in understanding the role of p53, study will send an important message to the community about possible background mutations that may eclipse some of their findings. Also, future efforts to identify the mutation(s) will become important as it is able to differentially suppress the life span benefits of DR.

**Ref:**

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mutations. Accepted in Plos One, Published on bioRxiv doi: https://doi.org/10.1101/2020.07.09.195503

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