Nutrition

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Vitamin B6 is essential for *C. elegans* development

Vitamins and other nutrients sustain systemic functions and development. However, the individual role of each macro- and micronutrient is still unknown. *Caenorhabditis elegans* is a good model for studying the effects of diet and nutrients on host processes, because it is possible to modify the genome of this nematode and the genome of its diet bacteria. A study in *Communications Biology* explores the role of bacterial vitamin B6 for *C. elegans* embryonic development.

Feeding *C. elegans* with *Lactiplantibacillus plantarum* or the standard feed *E. coli* OP50 arrested development in the former group, showing that different feed nutrient availability can produce a developmental effect. To ensure that a deficiency in ingesting the bacteria *L. plantarum* was not responsible for the developmental arrest in this group, the researchers fed fluorescent-stained *L. plantarum* to the worms, which showed that the bacteria were ingested and reached the gut. When growing in presence of *L. plantarum*, the worms showed starvation behavior, but later supplementation with *E. coli* reversed the developmental arrest, hinting that nutrients introduced later were responsible for the developmental recovery. *C. elegans* raised with abundant feeding on *L. plantarum* and trace amounts of *E. coli* completed their development, showing that trace amounts of some nutrients in *E. coli* were enough for development completion.

To further understand the metabolic pathways affected during developmental arrest, the team screened a library of *E. coli* mutants deficient for only one gene. Of the 29 mutants producing developmental arrest in *C.elegans*, five lacked genes related to vitamin B6 biosynthesis. From these 5 mutants that produced developmental arrest, the *pdxH* mutant, a gene involved in the last step to generate pyridoxal 5′-phosphate (PLP) the final metabolite of B6 synthesis, showed reversal of development arrest when the worms' diet was supplemented with PLP, showing that nutritional supplementation can rescue B6 synthesis. Additionally, *pdxH*-mutant-fed *C. elegans* showed altered expression of PLP-binding B6 metabolism-related genes, showing that *E. coli* vitamin B6 can regulate host PLP-binding activity.

These results show that the deficiency of a single vitamin can affect systemic functions. Given that metabolic pathways are evolutionarily conserved and *Lactiplantibacillus* is a commensal bacterium found in humans and other mammals, these results provide useful insights into host-microbiome relationships in mammals.

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