that provides modest positive selection for a gut-dwelling strain with the capacity to extract slightly greater energy from a key food source.

It is also possible, however, that the selection pressure could in some cases operate at higher levels (6). An individual host’s microbiome is not automatically inherited by its offspring. Although gut microbial communities are assembled anew in each individual, preliminary studies suggest that some strains are inherited from parents or siblings and then maintained for years or decades (7). Stochastic forces must also shape these microbial communities on an individual level, given that even monozygotic twins share only a limited set of microbial taxa (8). However, in the context of microbial sharing through some form of grooming or ingestion of small quantities of feces, the benefit could extend more widely. In a clinical setting, molecular analysis of fecal microbiota transplants between humans has shown that conspecific donor strains are more likely to durably colonize (9). Thus, a symbiotic advantage conferred by a microbial strain to an individual might extend to a larger subgroup of the population.

Similarly, across time, if a group of animals are forced to move to a new area and/or eat alternative foods during periodic times of hardship, there may be a selective advantage to retaining “bet-hedging” microbial community diversity across the animal population. Thus, although symbiosis is often experimentally considered as a single microbial strain conferring a singular benefit to a host, with this baseline data, one could also begin to explore evolutionary signatures of symbioses that extend from microbial strains to communities and from individual animals to animal populations.

Moeller et al.’s study once again underscores that hominids are multispecies superorganisms. It opens the door to investigations of the genetic features upon which fundamental host-microbe symbiotic relationships are based.

REFERENCES
Self-destruct button?

Paternal mitochondrial DNA, across species, is eliminated during fertilization, as illustrated below for the worm *C. elegans*. A model for the underlying mechanism involves disruption of paternal mitochondrial membranes.

![Diagram showing the process of fertilization and the role of paternal mitochondria](https://example.com/diagram.png)

**Sperm**

**Zygote**

**Oocyte**

**Fertilization**

**Pronuclei fusion during fertilization to form a zygotic nucleus. Both paternal and maternal mitochondria are present initially.**

**Mitochondria in the zygote, the paternal mitochondria degrade.**

1. CPS-6/ENDOG
2. Mitochondrial DNA (mtDNA)
3. Loss of membrane potential activates the mitophagy machinery, so how paternal mitochondria that was observed by Zhou et al. in genetic crosses with *C. elegans* cps-6 mutants.

An awareness of the interplay between mitochondrial and nuclear genes might influence our understanding of mutations in mitochondrial DNA that cause debilitating diseases, including those that affect optic nerves, muscles, and metabolism. U.S. and UK science and ethics panels gave limited approval to preventing these diseases by in vitro fertilization with nuclear transfer using genetic material from three parents: sperm from the father, an oocyte nucleus from the mother, and oocyte cytoplasm from a second female with healthy mitochondrial DNA. Recent experiments to test the effectiveness of this approach have, however, uncovered difficulties with heteroplasmy (heterogeneous population of mitochondrial DNA) (11). Small amounts of residual mutant mitochondrial DNA sometimes compromise the larger population of healthy mitochondrial DNA through genetic drift (12). Further mechanistic studies of mitochondrial inheritance may help solve this problem.

REFERENCES AND NOTES


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Demystifying the demise of paternal mitochondrial DNA
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