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Inducing rapid and slowed brain aging through manipulation of social tasks in honeybees

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The honeybee model features extremely diverse longevity patterns among castes of queens and workers. The phenotypic disparities between the longest-lived caste (queens) and shorter-lived workers are relatively fixed in early development. In contrast, flexible longevity patterns are found in the different worker types. In brief, workers pass through a sequence of worker-type specific social behaviors. These include nursing the brood (nurse bees), foraging for nectar and pollen (forager bees), and temperature regulation in winter (winter bees). Here we exemplify the dramatic consequences of worker-type differentiation for behavioral and cellular aging: rapid senescence in foragers is contrasted by a slowed aging progression in nurses, and by an apparent absence of aging symptoms (negligible senescence) in winter bees. By manipulating the social environment of bees we show how individuals with short and extremely long lifespan can be transformed into one another. Our behavioral, anatomical, proteomic and epigenomic screening data support that worker type transformations affect common symptoms of behavioral and cellular senescence in the brain. Among these symptoms are reduced learning function as well as changed lipofuscin and protein abundance, e.g. of synapse- and glia-specific proteins. We also found that cellular senescence differs between different tissues and brain regions. On the colony-level we identify brood load as a key regulator of aging. On the cellular level, we argue that the alternative utilization of a common yolk precursor protein (vitellogenin) in nursing and somatic maintenance can link social resource transfers with slowed aging. Such a role in somatic maintenance is supported by the presence of vitellogenin in unexpected cellular locations, for example in certain brain cells.