Sodium Benzoate, a Common Preservative, Inhibits Growth, Shortens Lifespan, Induces Premature Aging, and Accelerates Neurodegeneration

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Sodium benzoate is one of the most commonly used preservatives in the food industry. Although the compound is recognized as safe by the FDA, the effects of sodium benzoate on human health have been of interest to both the public and the scientific community. The nematode Caenorhabditis elegans (C. elegans) is an ideal model organism to study the health effects of sodium benzoate because of its simplicity and its well established genetic toolkit. In this study, I found that sodium benzoate restricts C. elegans growth, shortens its lifespan, induces premature aging, and accelerates neurodegeneration. Sodium benzoate functions in parallel with the insulin/IGF-1 signalling pathway to decrease lifespan. Using an Alzheimer's disease model that expresses human beta amyloid peptides, sodium benzoate was revealed to also significantly accelerate neurodegeneration. Sodium benzoate was also revealed to induce age-pigments in young worms by accumulating age-pigments in lysosome-related organelles (LROs). This novel discovery may be an explanation for the premature aging and accelerated neurodegeneration. Using GFP marker strains and quantitative RT-PCR assays, I uncovered the role of sodium benzoate in suppressing the irg-1 innate immunity gene expression. The compromised innate immunity response is another underlying mechanism for the phenotypes described above. Overall, these results reveal the long term detrimental effects of sodium benzoate on animal health and indicates that it may have similar consequences on human health.

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