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Abstract:	Sir2, an evolutionarily conserved NAD+-dependent deacetylase, has been implicated as a key factor in mediating organismal life span. However, recent contradictory findings have brought into question the role of Sir2 and its orthologs in regulating organismal longevity. In this study, we report that Drosophila Sir2 (dSir2) in the adult fat body regulates longevity in a diet-dependent manner. We used inducible Gal4 drivers to knock down and overexpress dSir2 in a tissue-specific manner. A diet-dependent life span phenotype of dSir2 perturbations (both knockdown and overexpression) in the fat body, but not muscles, negates the effects of background genetic mutations. In addition to providing clarity to the field, our study contrasts the ability of dSir2 in two metabolic tissues to affect longevity. We also show that dSir2 knockdown abrogates fat-body dFOXO-dependent life span extension. This report highlights the importance of the interplay between genetic factors and dietary inputs in determining organismal life spans. Using Drosophila, Kolthur-Seetharam and colleagues show that dSir2 in the metabolically relevant tissue of the adult fat bod y affects organismal life span in a diet-dependent manner. Although fat-body-specific dSir2 knockdown abolishes dietary restriction (DR)-mediated life-span extension, its overexpression mimics the effect of DR on longevity. The dSir2-dependent longevity phenotype is limited to conditions that lead to an increase in both its own expression and NAD+ levels. The data also indicate that fat-body dSir2 is indispensable for dFOXO-dependent life-span extension.
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