

Is diabetes protective against amyotrophic lateral sclerosis?

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A study of patients in Denmark suggests that type 2 diabetes may be associated with a reduced risk for the fatal neurodegenerative disease amyotrophic lateral sclerosis (ALS), according to an article published online by *JAMA Neurology*.

Recent reports have suggested a protective association between [vascular risk factors](#), such as obesity or higher body mass index (BMI), higher cholesterol levels and hyperlipidemia with ALS incidence and survival. Patients with type 2 diabetes have, on average, higher BMI, elevated blood lipid levels and defective energy metabolism. However, the association between diabetes and ALS has not been widely explored.

Marianthi-Anna Kioumourtzoglou, Sc.D., of the Harvard T.H. Chan School of Public Health, Boston, and coauthors, examined the association between diabetes, obesity and ALS using data from Danish National Registers for 3,650 patients diagnosed with ALS between 1982 and 2009. The average age at diagnosis was 65.4 years. They were compared with 365,000 healthy control patients.

The authors also identified 9,294 patients with diabetes at least three years prior to the index date (the date of ALS diagnosis or the same date for the matched controls), 55 of whom were subsequently diagnosed with ALS. The average age of the first diabetes-related diagnosis was 59.7 years.

The study found that diabetes, but not obesity, was associated with a reduced risk of ALS. The association with diabetes was affected by both age at ALS diagnosis and age at diabetes diagnosis, with older age at diagnosis for either disease associated with lower risk for ALS.

"We conducted a nationwide, population-based study and observed an overall protective association between diabetes and ALS diagnosis, with the suggestion that type 2 diabetes is

protective and type 1 [diabetes](#) is a risk factor.

Although the mechanisms underlying this association remain unclear, our findings focus further attention on the role of [energy metabolism](#) in ALS pathogenesis," the study concludes.

More information: *JAMA Neurol.* Published online June 1, 2015. [DOI: 10.1001/jamaneurol.2015.0910](#)

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