

Oral α -lipoic acid improves symptoms of DSP in patients with diabetes

Distal symmetric polyneuropathy (DSP) is common in patients with diabetes and causes severe neuropathic pain that reduces quality of life. Antioxidants improve the underlying pathophysiology of DSP, so Ziegler *et al.* evaluated the efficacy and dose-response characteristics of the antioxidant α -lipoic acid (ALA) in this setting.

In a multicenter, randomized, double-blind, placebo-controlled trial, the authors evaluated 181 patients (aged 18–74 years) with diabetes and DSP from Russia and Israel. All patients received placebo once daily for 1 week, and were then randomly allocated to receive placebo ($n=43$), ALA 600 mg ($n=45$), ALA 1,200 mg ($n=47$), or ALA 1,800 mg ($n=46$) orally once daily for an additional 5 weeks. Patients' total symptom scores were assessed at baseline and weekly thereafter.

At study end there was a marked reduction in the mean total symptom score, and in its subscores for lancinating and/or stabbing and burning pain (but not for paresthesia or numbness) in the ALA-treated groups, compared with the placebo-treated group. Significant improvements were observed after 1–2 weeks for all ALA-treated patients ($P<0.05$). Treatment with ALA 600 mg produced the least gastrointestinal adverse effects.

The effects of ALA on DSP were not dose-dependent, so the authors recommend once-daily administration of oral ALA 600 mg. The magnitude of the effect on DSP symptoms at this dose was similar to that of the previously reported effect of intravenous ALA 600 mg over 3 weeks.

Original article Ziegler D *et al.* (2006) Oral treatment with α -lipoic acid improves symptomatic diabetic polyneuropathy. *Diabetes Care* 29: 2365–2370

Coffee drinkers have a reduced risk of developing type 2 diabetes

Coffee consumption has been suggested to reduce the risk of developing type 2 diabetes. Previous studies, however, mostly relied on self-reported diabetes. Smith and colleagues examined a white Californian community

to assess the association between coffee drinking and incident diabetes—defined as no history of diabetes at baseline with subsequent onset of diabetes confirmed by oral glucose tolerance tests.

In total, 910 adults (mean age 65.9 years) were followed for 8 years on average after assessment of coffee intake. Data from 593 participants with normal baseline glucose levels and 317 participants with impaired baseline glucose levels were analyzed separately. Of the 910 participants, 97 never drank coffee, 153 were past coffee drinkers and 660 were current coffee drinkers (who consumed 2.8 cups per day on average). The risk of type 2 diabetes was approximately 60% lower in current and past coffee drinkers (odds ratios 0.36 and 0.38, 95% CI 0.19–0.68 and 0.17–0.87, respectively) than in individuals who never drank coffee. This effect was similar in participants with normal or impaired baseline glucose levels and was independent of age, sex, exercise, BMI, smoking, alcohol consumption and hypertension.

The authors discuss the possible physiological mechanisms of the protective effect of coffee and the geographical differences in terms of the contents of the coffee consumed. The component of coffee responsible for its favorable effect on the risk of diabetes, however, remains to be investigated.

Original article Smith B *et al.* (2006) Does coffee consumption reduce the risk of type 2 diabetes in individuals with impaired glucose? *Diabetes Care* 29: 2385–2390

Short-term lifestyle intervention reduces the long-term risk of type 2 diabetes

The randomized, controlled Finnish Diabetes Prevention Study (FDPS) revealed that lifestyle intervention prevented or postponed progression to type 2 diabetes, in high-risk individuals, while intervention continued. Lindström and colleagues now report the results of an extended follow-up of participants in the FDPS who did not develop diabetes during the original study.

The FDPS enrolled 522 overweight individuals (mean age 55 years) with impaired glucose tolerance, who were randomly allocated to receive either ongoing, tailored lifestyle intervention for a median of 4 years, or nonindividualized information on health-modifying behavior