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File: ■ Olive (*Olea europaea*, Oleaceae) Oil
■ Dietary Fat
■ Postprandial Glycemic Response
■ Type 1 Diabetes Mellitus

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RE: Olive Oil Reduces Postprandial Glycemic Response in Patients with Type 1 Diabetes

Bozzetto L, Alderisio A, Giorgini M, et al. Extra-virgin olive oil reduces glycemic response to a high-glycemic index meal in patients with type 1 diabetes: a randomized controlled trial. *Diabetes Care*. April 2016;39(4):518-524.

Postprandial glycemic response is an important factor in controlling blood glucose in persons with type 1 diabetes. Although carbohydrate content of a meal is considered the main dietary factor influencing postprandial glycemia, growing evidence suggests the fat content of a meal also influences glycemic response. These authors conducted a randomized, crossover study in patients with type 1 diabetes to test the hypothesis that monounsaturated fat from extra virgin olive (*Olea europaea*, Oleaceae) oil (EVOO) would reduce postprandial glycemic response.

Thirteen patients with type 1 diabetes (8 women and 5 men) were recruited from the diabetes care unit of the Federico II University teaching hospital in Naples, Italy. Inclusion criteria included treatment with continuous subcutaneous insulin infusion, the use of fast-acting insulin analogs for at least 6 months, and a glyated hemoglobin (HbA1c) <8.0%. The mean age of the patients was 38 ± 11 years; body mass index was 24.8 ± 2.9 kg/m². Duration of diabetes was 25 ± 3 years. The total daily insulin dose among the patients was 41.1 ± 10.7 IU; they had acceptable blood glucose levels.

Before the start of the study, the patients took part in a 1-week run-in period during which they underwent continuous glucose monitoring (CGM) and completed a 7-day dietary record to optimize basal infusion rate and insulin-to-glycemic load ratio. The patients were randomly assigned to a 1-week period during which they consumed either 3 high-glycemic index (HGI) meals or low-glycemic index (LGI) meals. They then crossed over to the alternative meals for 1 week. The HGI and LGI meals were similar in total carbohydrate content but differed in amount and type of fat and were categorized as low in fat (low-fat), high in saturated fat (butter), or high in monounsaturated fats from EVOO. During the 2 weeks of the study, the patients wore sensors at all times for CGM. The patients checked capillary blood glucose at 2, 4, and 6 hours after the test meals.

The study procedures were the same for both test weeks. The 3 test meals were eaten at

lunch on days chosen according to the patients' work and recreational activities to keep these activities reproducible and compatible with the study design. On the mornings of the test meal days, the patients ate the same light breakfast to avoid a second-meal effect bias. They avoided strenuous physical activity on the day before the test meal, the morning of the test meal, and for 6 hours after the meal. Pre-meal insulin doses, which were based on the insulin-to-glycemic load ratio determined for each patient, were significantly lower before the LGI meals compared with doses administered before the HGI meals ($P < 0.0001$).

The EVOO and butter meals were similar in energy content; the low-fat meal had a lower energy content. The glycemic index was about 25% greater in the HGI meals than in the LGI meals. Dietary fiber was greater (by about 13 g) in the LGI meals compared with the HGI meals.

The HGI meals included white rice (*Oryza sativa*, Poaceae) (60 g), white bread (75 g), minced beef (90 g), and banana (*Musa paradisiaca*, Musaceae) (180 g), plus butter (43 g) or EVOO (37 g). The LGI meals included pasta (50 g), lentils (*Lens culinaris*, Fabaceae) (100 g), whole-meal bread (30 g), ham (15 g), and apple (*Malus pumila*, Rosaceae) (185 g), plus butter (45 g) or EVOO (37 g).

The authors report that the 6-hour postprandial glucose profile was significantly different between HGI and LGI meals ($P=0.005$), being significantly higher during the first 3 hours after the HGI meals with a tendency to an opposite pattern later. Although the time to glucose peak was significantly delayed after LGI compared with HGI meals ($P=0.003$), no significant differences were observed in the peak values between the LGI (4.7 ± 0.7 mmol/L) and HGI (5.3 ± 0.9 mmol/L) meals. The quality and amount of fat in the LGI meals did not significantly influence postprandial blood glucose response, blood glucose peak, or time to glucose peak.

With the HGI meals, postprandial blood glucose was significantly lower after EVOO than after low-fat or butter meals ($P<0.0001$), with a marked difference from baseline to 3 hours between EVOO and either low-fat or butter ($P<0.05$) meals. The blood glucose peak was lower, although not significantly, after the EVOO meal than after the butter or low-fat meals. The time to blood glucose peak was significantly delayed after the EVOO meal (190 ± 101 minutes) compared to after the butter (188 ± 104 minutes) or low-fat (146 ± 81 minutes) meal ($P=0.035$).

In this study, the type of fat significantly influenced the postprandial glycemic response in patients with type 1 diabetes. The addition of different types of fats to meals with an LGI did not influence postprandial blood glucose response; however, different types of fats added to HGI meals did influence the response. These results, which indicate that the combination of carbohydrate foods and type of fat should be considered when determining the timing and dose of prandial insulin administration, have important clinical implications for persons with type 1 diabetes. A possible weakness of this study is the consumption of meals at the patient's home without direct supervision, which could have affected the standardization of procedures. The home setting also limited the gathering of information on possible mechanisms responsible for the results.

—*Shari Henson*

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