and again at 18 months (p<0.001). There was a significant decline in median stimulated C-peptide concentrations for both groups from baseline to Month 12 (0.88 to 0.73 nmol/l for atorvastatin and 0.89 to 0.71 nmol/l for placebo; p<0.01 for both). This decline continued in the placebo group at Month 18 (p=0.047) but did not occur in the atorvastatin group.

The course of metabolic control was similar between the two groups. However, at 12 months, insulin doses varied between the two groups (p=0.007). An increase in HbA1C levels was observed in the placebo group from 12 to 18 months, but this was not the case in the atorvastatin group. Those who were treated with atorvastatin demonstrated decreases in systemic levels of C-reactive protein, total and low-density lipoprotein cholesterol, and triglycerides (p<0.001 for all). Additionally, the course of the chemokine MCP-1 differed between the two groups, as a decrease was observed in the placebo group but not in the atorvastatin group (p=0.009). The course of the other 14 immune mediators appeared to be similar for atorvastatin and placebo. There did not appear to be covariates that were associated with atorvastatin and C-peptide secretion such as age, body mass index, or baseline serum C-peptide concentrations.

Atorvastatin may delay the loss of residual beta-cell function in patients with newly diagnosed T1DM and merits further investigation in a larger clinical trial.

Caffeine Supplementation for the Prevention of Exercise-Induced Hypoglycemia

Caffeine ingestion prior to exercise may reduce hypoglycemic episodes and slow the decline in blood glucose during exercise in patients with type 1 diabetes mellitus (T1DM). Ian W. Gallen, MD, Wycombe Hospital, Buckinghamshire, United Kingdom, presented findings from a small study that investigated the physiological impact of caffeine ingestion prior to exercise.

This study evaluated 5 patients with T1DM (4 males and 1 female) who were on multiple daily injection therapy. The mean age was 38.2 years, and the mean body mass index was 27.32. Study Day 1 consisted of workload assessment, as determined by maximum oxygen consumption (VO_2 max). Patients were randomized to receive either caffeine 5 mg/kg or placebo 2 hours postprandially/after glargine-aspart injection with rest for 30 minutes prior to exercise. Subjects exercised on a cycle ergometer for 10 minutes at 50% VO_2 max, increasing to 70% VO_2 max for 30 minutes. After a total of 40 minutes of exercise, patients rested for 30 minutes. Arterialized blood glucose and lactate were measured every 10 minutes, gas exchange was measured continuously, and respiratory exchange rate (RER) was calculated. At least 1 week later, an identical exercise protocol was administered, but the patients received the opposite treatment regimen (those who previously received caffeine were given placebo and vice versa).

Blood glucose remained similar to baseline levels throughout the duration of the exercise and into the resting phase of the program in those who received caffeine. None of the subjects in the caffeine group required oral glucose. Subjects who were taking caffeine demonstrated significantly higher blood glucose levels compared with placebo after exercising at 70% VO_2 max for 10 minutes (p=0.014), after exercising at 70% for 30 minutes (p=0.032), and after 30 minutes of postexercise rest (p=0.01). Blood glucose levels began to fall at the start of exercise in those who received placebo, and this rapid decline continued throughout the exercise program. Oral glucose (20 g) was administered to 2 of the patients in the placebo group in order to avoid a hypoglycemic event.

Lactate and RER measurements remained relatively consistent throughout the exercise program in those who received caffeine. Caffeine did not appear to alter RER or increase lactate levels, which suggests that caffeine does not alter substrate oxidation. These findings also suggest that caffeine may augment endogenous glucose production and oxidation when coupled with exercise.

Caffeine may be a useful tool in the prevention of hypoglycemic events during exercise in patients with T1DM. However, larger studies are needed to establish the safety and efficacy of such an approach.