Abstract

The contribution of fat oxidation to energy production during exercise is influenced by intensity of exercise. The aim of this study was to assess the relationship between the highest value of fat oxidation rate (FATmax) and the oxygen uptake (VO2) in sedentary type 2 diabetes (T2D) patients vs healthy sedentary subjects. Sedentary T2D patients and healthy sedentary subjects were evaluated to a graded exercise test, and oxygen uptake and fat oxidation rate were detected. Data show that in T2D patients fat oxidation rate is not impaired and the positive linear correlation between FATmax and both VO2 and VO2max suggests that even in T2D patients the muscle oxidative capacity might increase in response to aerobic training.

Introduction

Carbohydrate and fatty acids are the dominant fuels oxidized for energy production during exercise. Their contribution is influenced by intensity of exercise, with a progressive increase in the relative contribution of carbohydrate oxidation to energy expenditure, whereas the absolute rate of fat oxidation increases from low to moderate intensities of exercise and declines as exercise becomes more intense. In patients with type 2 diabetes (T2D) the altered metabolism and the lower capacity to sustain efforts changed the choice and regulation of energy substrates for oxidation.

Purpose of this study was to assess the relationship between the highest value of fat oxidation rate (FATmax) and the oxygen uptake (VO2) in sedentary T2D patients in comparison with healthy sedentary subjects.

Materials and Methods

Fifteen sedentary T2D patients (9 males, 6 females), mean age 58 (range 39-65) and fifteen healthy sedentary subjects (8 males, 7 females), mean age 53 (range 37-71), were studied. The physical and physiological characteristics of subjects are shown in Table 1.

All T2D patients were being treated with diet and antiglycemic agents. Significant cardiovascular disease was excluded in all subjects by an instrumental examination. Each subject gave his written informed consent after explanation of the experimental procedures. The exercise testing started in the laboratory at 8.00 a.m. after an overnight fast. All subjects performed a graded exercise test to exhaustion on treadmill, using modified Bruce protocol. Oxygen uptake (VO2) and carbon dioxide (VCO2) were recorded with a breath-by-breath measurement system (Cosmed Quark CPET; Cosmed Srl, Rome, Italy) and maximum fat oxidation rate (FATmax) was determined using indirect calorimetry, with the assumption that urinary nitrogen excretion rate was negligible. Peak VO2 was recorded as the highest consecutive 30 s average value during the last minute of exercise or early recovery. Substrate oxidation rates were then plotted as a function of exercise intensity, expressed as percentage of maximal oxygen uptake (VO2max).

From each fat oxidation curve, the peak rate of fat oxidation measured over the entire range of exercise intensities (FATmax and the exercise intensity at which the fat oxidation rate was maximal, in absolute and percentage of maximal oxygen uptake (VO2; %VO2max), were identified. All responses were monitored throughout rest, exercise and recovery, and graphically displayed. Data are shown as means±standard deviation.

Results

The average of body mass index (BMI) was 28.7±1.07 for T2D patients and 26.6±1.3 for healthy subjects. The average VO2max was 22.29±1.06 and 28.63±2.03 mL/kg/min for T2D patients and healthy subjects, respectively (P<0.01). In T2D patients, absolute fat oxidation rate reached a maximum of 6.71±0.46 mg/kg/min at VO2 of 15.41±0.68 mL/kg/min (70±1.27% of VO2max), while in healthy subjects 7.19±0.77 mg/kg/min at VO2 of 18.23±1.28 mL/kg/min (64±2.61% of VO2max). A positive linear correlation (r=0.85; P=0.0001) between FATmax and VO2 in both groups has been found (Figure 1).
Also, positive linear correlation between FAT_max and VO2max in both type 2 diabetes (r=0.76; P=0.001) and healthy subjects (r=0.73; P=0.002) has been found (Figure 2).

Discussion

Exercise intensity looks very important in determining substrate utilization. In normal subjects, on increasing exercise intensity from low to moderate, absolute lipid oxidation keeps increasing but, on increasing further, fat oxidation rates decrease markedly. Subjects with T2D could have an abnormal pattern of substrate recruitment for oxidation during exercise. Our data, as found in literature, indicate that in T2D patients fat oxidation rate is not impaired. VO2max, index of exercise capacity, was significantly lower in T2D patients so that, in T2D compared to healthy subjects, we found a maximal fat rate oxidation at higher exercise inten-
Moreover, the positive linear correlation between $\text{FAT}_{\text{max}}$ and both $\text{VO}_2$ and $\text{VO}_2\text{max}$ suggests that even in T2D patients the muscle oxidative capacity might increase in response to aerobic training, and this could contribute to reduction of body fat mass.

References