Cardiac lipid content and systolic function in overweight type 2 diabetic subjects after exercise training

V. Schrauwen-Hinderling[1], R. Meex[1], J. Glatz[1], J. Wildberger[1], H. Lamb[2], M. Schär[3,4], M. Hesselink[1], P. Schrauwen[1], and M. E. Kooi[1]

[1]Maastricht University Medical Center, Maastricht, Netherlands, [2]Leiden University Medical Center, Leiden, Netherlands, [3]Johns Hopkins University, Baltimore, United States, [4]Philips Health Care, Cleveland, United States

Background: Excessive lipid storage in cardiac muscle has been suggested to hamper cardiac function via 'lipotoxic' pathways. We reported earlier that physical activity training in healthy overweight subjects improves systolic function and diminishes cardiac lipid content. It is yet unknown whether diabetic subjects respond similarly.

Objective: To investigate whether physical activity improves cardiac function and decreases cardiac lipid content in type 2 diabetic subjects.

Subjects and methods: Eleven overweight male patients with type 2 diabetes mellitus (age: 59.5±0.9years, BMI: 30.5±1.3kg/m^2, fasting plasma glucose: 9.1±0.6mmol/l, HbA1c: 7.1±0.3%) followed a supervised 12-week physical activity training program (three sessions/week). Before and after training, maximal oxygen uptake (VO_2max) was measured, systolic function was determined by CINE-MRI (Slice thickness = 6 mm, gap = 4 mm (flip angle = 50°; shortest possible TR (3.3 ms) and TE (1.67 ms); field of view = 350 x 350 mm, reconstructed matrix = 256 x 256, number of heart phases = 24, Intera, 1.5T, Philips Healthcare), cardiac lipid content was determined by image–guided Magnetic Resonance Spectroscopy with a PRESS-sequence (TE = 26ms; TR = 4s), ECG-triggered and respiratory-gated with pencil beam navigator. High efficiency of the navigator-gated acquisition was achieved by instructing patients to breathe in the rhythm of the measurement.

Results: VO_2max and ejection fraction were improved after training (VO_2max: from 27.1±1.5ml/min/kg to 30.1±1.6ml/min/kg (p=0.001), EF from 50.5±2.0% to 55.6±1.5% (p=0.001)). Surprisingly, cardiac lipid content did not decrease with training (from 0.80±0.07% to 0.96±0.07%, p=0.15 (intensity of CH_2-resonance of lipids relative to water-resonance)).

Conclusion: While the currently employed training program increased cardiac function in this group of diabetic subjects, it did not affect cardiac lipid content. This is in contrast to our earlier findings in healthy overweight subjects, where the same training intervention diminished cardiac lipid content. These observations may indicate hampered exercise-induced lipid mobilization in the diabetic heart and reveal that reduction of cardiac lipids is not a prerequisite for the training-induced improvement in cardiac function.

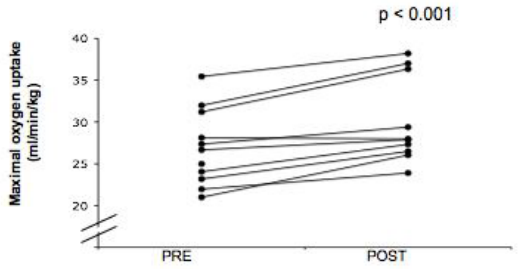

Figure 1: Whole body maximal oxygen uptake is increased by 11% after physical exercise training.

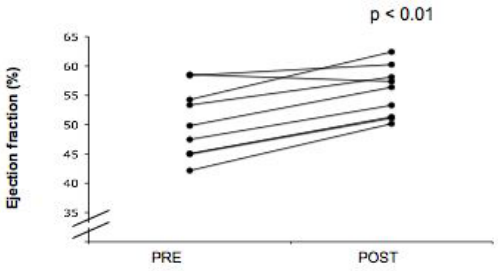

Figure 2: Ejection fraction is improved by 10% after the training program (n=9).

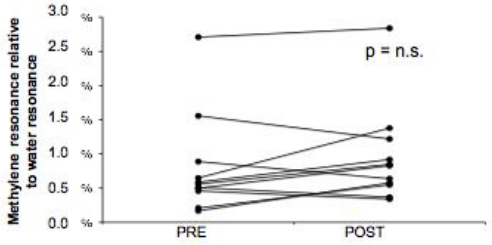

Figure 3: Unchanged cardiac lipid content after training.