
In this study, the authors assessed whether organ-specific fat accumulation and cardiac function in Type 2 diabetes is affected by exercise intervention. In total, 12 individuals with Type 2 diabetes (seven men; average age 46 ± 2 years) had their fat volume measured by magnetic resonance imaging at baseline and after 6 months of moderate-intensity exercise, followed by a high-altitude trekking expedition with exercise of long duration. Cardiac function was also quantified in these individuals. It was observed that although subcutaneous abdominal fat volume did not change (p = 0.9), visceral abdominal fat volume was reduced from 348 ± 57 to 219 ± 33 ml (p < 0.01) by exercise. Furthermore, hepatic intramyocellular triglyceride content and pericardial fat volume were also decreased by exercise from 6.8 ± 2.3 to 4.6 ± 1.6% (p < 0.01) and from 4.6 ± 0.9 to 3.7 ± 0.8 ml (p = 0.02), respectively. However, cardiac function did not change with exercise (p = 0.03). Overall, the study suggested that in individuals with Type 2 diabetes, a 6-month exercise intervention can induce tissue-specific changes in body fat distribution, which could lower cardiovascular risk.


The authors used a subcohort from the HART-D study to evaluate the determinants of exercise training-induced improvements in glucose control in Type 2 diabetics. A total of 35 individuals were recruited (17 men and 18 females, aged 57.0 ± 7.7 years) and assessed using muscle biopsies at baseline and after 9 months of intervention (aerobic, resistance or combination training). It was observed that there was an association between changes in glucose control and adiponectin levels (r = -0.45; p = 0.007) and the largest increase in skeletal muscle PGC-1α was observed in individuals who had a longer duration of Type 2 diabetes (r = 0.44; p = 0.008). The results suggested that approximately 65% of the variability in the changes in glucose control are explained by male sex (p = 0.05), non-Caucasian ethnicity (p = 0.02), duration of Type 2 diabetes (r = 0.40; p < 0.002) and changes in free fatty acids (r = 0.36; p < 0.004), adiponectin (r = -0.26; p < 0.03) and skeletal muscle PGC-1α (r = -0.28; p = 0.02). However, the authors did observe that their investigation was limited by a small sample size, of which almost half the individuals were in the resistance training group.

Using participants from the Adult Changes in Thought study, the authors evaluated whether there is an increased risk of dementia in nondiabetics who have higher glucose levels. In total, 2067 individuals (232 diabetics and 1835 nondiabetics; 839 men and 1228 women) without dementia were assessed using clinical measurements of glucose and HbA1c levels. It was observed that dementia developed in 524 individuals (74 diabetics and 450 nondiabetics) at a median follow-up of 6.8 years. In the nondiabetic group, there was an increased risk of dementia in individuals who had higher average glucose levels (glucose level of 115 vs 100 mg/dl) within the preceding 5 years (p = 0.01); the adjusted hazard ratio for dementia was 1.18 (95% CI: 1.04–1.33). It was also observed that there was an increased risk of dementia in diabetic individuals who had higher average glucose levels (190 vs 160 mg/dl; p = 0.002); the adjusted hazard ratio was 1.40 (95% CI: 1.12–1.76). Overall, the results suggest that even in nondiabetic individuals, higher glucose levels could be a risk factor for dementia.

— Paper suggestions by Dominque Hansen (Hasselt University, Faculty of Medicine, Belgium). All stories written by Natasha Leeson.