

Author, Year; Country Score Research Design Sample Size	Methods	Outcomes
de Groot et al. 2003; Netherlands PEDro = 7 RCT Level 1 N = 6	<b>Population:</b> 4 male, 2 female, C5-L1, AIS A ( $n = 1$ ), B ( $n = 1$ ), and C ( $n = 4$ ), age 36 yrs, 116 d post-injury. <b>Treatment:</b> Randomized to low-intensity (50%–60% HRR) or high-intensity (70%–80% HRR) arm ergometry, 20 min/d, 3 d/wk, 8 wks. <b>Outcome Measures:</b> VO <sub>2</sub> peak, insulin sensitivity, blood glucose.	<ol style="list-style-type: none"> <li>1. There was a significant difference in insulin sensitivity between groups, with a non-significant decline in the high-intensity group and a significant improvement in the low-intensity group with training.</li> <li>2. A positive correlation between VO<sub>2</sub>peak and insulin sensitivity (<math>r = 0.68</math>, <math>p = 0.02</math>).</li> </ol>
Jeon et al. 2010; Canada Pre-post Level 4 N = 6	<b>Population:</b> 6 male participants with paraplegia participated in the study (mean age, $48.6 \pm 6.0$ y; mean weight, $70.1 \pm 3.3$ kg; injury levels between T4-5 and T10). <b>Treatment:</b> 12 weeks of FES-rowing exercise training 3 to 4 times a week (600–800 kcal). <b>Outcome measures:</b> VO <sub>2</sub> peak, plasma leptin, insulin, and glucose levels, insulin sensitivity, body composition.	<ol style="list-style-type: none"> <li>1. VO<sub>2</sub>peak increased from <math>21.4 \pm 1.2</math> to <math>23.1 \pm 0.8</math> mL·kg<sup>-1</sup>·min<sup>-1</sup> (<math>P = 0.048</math>).</li> <li>2. Plasma leptin levels were significantly decreased after the training (pre: <math>6.91 \pm 1.82</math> ng·dL<sup>-1</sup> vs. post: <math>4.72 \pm 1.04</math> ng·dL<sup>-1</sup>; <math>P = 0.046</math>).</li> <li>3. Plasma glucose and leptin levels were significantly decreased after exercise training by 10% and 28% (<math>P = 0.028</math>), respectively.</li> <li>4. Plasma glucose, Leptin levels and Whole body fat decreased but did not reach statistical significance.</li> </ol>
Mahoney et al. 2005; USA Pre-post Level 4 N = 5	<b>Population:</b> 5 males, complete SCI, C5-T10, AIS grade A, age 35.6 yrs, 13.4 yrs post-injury. <b>Treatment:</b> Home-based neuromuscular electric stimulation-induced resistance exercise training, 2 d/wk, 12 wks. <b>Outcome Measures:</b> quadriceps femoris muscle cross-sectional area, plasma glucose, insulin.	<ol style="list-style-type: none"> <li>1. All participants had normal fasting glucose levels before and after training.</li> <li>2. There were no significant changes in blood glucose or insulin with training. However, there was a trend towards reduced plasma glucose levels (<math>p = 0.074</math>).</li> </ol>
Phillips et al. 2004; Canada Pre-post Level 4 N = 9	<b>Population:</b> 8 male, 1 female, incomplete AIS C, C4-T12, 8.1 yrs post-injury. <b>Treatment:</b> Body-weight-supported treadmill walking, 3 d/wk, 6 months. <b>Outcome Measures:</b> whole-body dual-energy X-ray absorptiometry (to capture body composition and bone density), GLUT4 protein abundance, hexokinase activity, oral glucose tolerance tests, glucose oxidation, CO <sub>2</sub> breath analysis.	<ol style="list-style-type: none"> <li>1. Reduction in the area under the curve for glucose (-15%) and insulin (-33%).</li> <li>2. The oxidation of exogenous (ingested) glucose and endogenous (liver) glucose increased (68% and 36.8%, respectively) after training.</li> <li>3. Training resulted in increased muscle glycogen, GLUT-4 content (glucose transporter) (126%), and hexokinase II enzyme activity (49%).</li> </ol>
Jeon et al. 2002; Canada Pre-post Level 4 N = 7	<b>Population:</b> 5 male, 2 female, motor complete, C5-T10, ages 30-53 yrs, 3–40 yrs post-injury. <b>Treatment:</b> FES leg-cycle training, 30 min/d, 3 d/wk, 8 wks. <b>Outcome Measures:</b> oral glucose tolerance test (OGTT), glucose and insulin levels, glucose utilization, insulin sensitivity and levels.	<ol style="list-style-type: none"> <li>1. There were significantly lower (14.3%) 2-hr OGTT glucose levels after 8 wk of training.</li> <li>2. Glucose utilization was higher for all 3 participants and insulin sensitivity was higher for 2 of the 3 participants during posttraining 2-hr clamp test.</li> </ol>

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<p>Mohr et al. 2001; Denmark Pre-post Level 4 N = 10</p>	<p><b>Population:</b> 8 male, 2 female, 6 tetraplegia, 4 paraplegia, C6-T4, age 35 yrs, 12 yrs post-injury. <b>Treatment:</b> FES cycling, 30 min/d, 3 d/wk, 12 months; 7 participants completed an additional 6 months (1 d/wk). <b>Outcome Measures:</b> insulin-stimulated glucose uptake, oral glucose tolerance test (OGTT), GLUT 4 glucose transporter protein.</p>	<ol style="list-style-type: none"> <li>1. Insulin-stimulated glucose uptake rates increased after intensive training.</li> <li>2. With the reduction in training, insulin sensitivity decreased to a similar level as before training. GLUT-4 increased by 105% after intense training and decreased again with the training reduction. The participants had impaired glucose tolerance before and after training, and neither glucose tolerance nor insulin responses to OGTT were significantly altered by training.</li> </ol>
<p>Chilibeck et al. 1999; Canada Pre-post Level 4 N = 5</p>	<p><b>Population:</b> 4 male, 1 female, motor complete C5-T8, ages 31–50 yrs, 3–25 yrs post-injury. <b>Treatment:</b> FES leg-cycle ergometry training, 30 min/d, 3 d/wk, 8 wks. <b>Outcome Measures:</b> glucose transporters (GLUT-4, GLUT-1), oral glucose tolerance test, citrate synthase.</p>	<ol style="list-style-type: none"> <li>1. Training resulted in increases in GLUT-1 (52%) and GLUT-4 (72%).</li> <li>2. There was a training-induced increase in citrate synthase activity (56%) and an improvement in the insulin sensitivity index as determined from oral glucose tolerance test.</li> </ol>
<p>Hjeltnes et al. 1998; Sweden Pre-post Level 4 N = 5</p>	<p><b>Population:</b> 5 males, C5-C7, all complete AIS A, age 35 yrs, 10 yrs post-injury. <b>Treatment:</b> Electrically stimulated leg cycling exercise, 7 d/wk, 8 wks. <b>Outcome Measures:</b> peripheral insulin sensitivity, whole body glucose utilization, glucose transport, phosphofructokinase, citrate synthase, hexokinase, glycogen synthase, blood glucose, plasma insulin.</p>	<ol style="list-style-type: none"> <li>1. After training, insulin-mediated glucose disposal was increased by 33%. There was a 2.1-fold increase in insulin-stimulated glucose transport.</li> <li>2. Training led to marked increases in protein expression of GLUT4 (glucose transporter) (378%), glycogen synthase (526%), and hexokinase II (204%) in the vastus lateralis muscle.</li> <li>3. Hexokinase II activity increased 25% after training.</li> </ol>

Note: AIS = ASIA Impairment Scale; d = day; FES = functional electrical stimulation; HRR = heart rate reserve; min = minute; OGTT = oral glucose tolerance test; RCT = randomized controlled trial; SCI = spinal cord injury; wk = week.