Lipid metabolism during exercise

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Outline

• Factors influencing lipid metabolism during exercise

• Effect of exercise intensity
  - Whole body vs. Limited muscle mass
  - Endogenous & Exogenous FA

• Fat oxidation in arm and leg muscle during exercise
Arbeitsfähigkeit und Ernährung

Von
E. Hohwü Christensen und Ove Hansen

1939!
(a)

Whole body fat oxidation, g min$^{-1}$

- Trained
- Untrained

Relative workload, % VO$_2$max

Time, min

Workload, Watt

RER > 1.0 finish test

Nordby et al., 2006

Achten et al. 2002
Substrate oxidation (% of energy)

FAT    CHO
Whole body

Carbohydrates

Carbohydrates

Helge et al. 2001
Horowitz et al. 1997
Factors influencing fat oxidation during exercise

- **Duration** of exercise
- **Exercise intensity**
- **Diet/supplement**
- **Training** status
- **Exercise mode**
- **Surroundings** (temp, altitude, etc.)
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Fat combustion
kcal min\(^{-1}\)

\[\text{VO}_2 \text{ l/ min} \]

Galbo, H., 1991
Van Loon et al. 2001

Energy expenditure (kJ min\(^{-1}\))

- **Other fat sources**
- **Plasma FFA**
- **Plasma glucose**
- **Muscle glycogen**

Exercise intensity (%Wmax)
Attenuation of lipid oxidation at high exercise intensity

1. Decreased activation of FA mediated via a down regulated activity of acyl-CoA synthase by increased AMP and/or decreased CoASH

2. Decreased transport of long chain FA into mitochondria via the CPT system. This could be down regulated through a decreased muscle PH and/or depletion of free carnitine

3. Decreased fat oxidation due to feed-back control by redox state on β-oxidation. The difference in redox equivalents feeding into complex I (CHO) or complex I & II (FAT) may theoretically provide a means of regulation at the ETC

- Regulation of FA transport at the level of the sarcolemma is at present not considered of major importance
Investigate the effect of exercise intensity on substrate utilization during one-leg exercise
Nine healthy male: 25 ± 1 yr., 76 ± 3 kg, 182 ± 2 cm; $\text{VO}_{2\text{max}}$ 3.9 ± 0.2 l O$_2$ min$^{-1}$

NaH$_{13}$CO$_2$ prime
Infusion [U-13C-palmitate]
(0.015 µmol kg$^{-1}$ min$^{-1}$)

One-leg kicking with both legs
25 % Wmax
25 % Wmax
85% Wmax
65% Wmax

Rest:
Blood samples femoral arterial & venous (both legs) and blood flow (thermodilution)

Muscle biopsy vastus lateralis (both legs)

Helge et al. 2007
Leg Fat oxidation, µmol min\(^{-1}\)

Exercise intensity, % Wmax

- Rest
- 25
- 65
- 85

Plasma FA oxidation

Non plasma FA oxidation

* * *

Helge et al. 2007
Substrate oxidation,

Oxygen equivalents, ml O₂

Exercise intensity,
% Wmax

Plasma FA oxidation

Glycogen

Plasma glucose

Leg oxygen uptake

Helge et al. 2007
Plasma FA (µmol l\(^{-1}\))

687 57, 720 111, 719 110

(25) (65) (85)

FA delivery, µmol min\(^{-1}\)

Lactate release, mmol min\(^{-1}\)

Helge et al. 2007
One set of slides (6 in total) containing an unpublished experiment is taken out of the presentation
% $\text{VO}_2\text{max}$

- 65 con. 43 $\mu\text{mol/kg/min}$
- 85 con. 27 3 $\mu\text{mol/kg/min}$
- 85 lipid 34 5 $\mu\text{mol/kg/min}$

Diagram A: Plots of plasma FFA (mM) over time (min) for Lipid/Heparin Infusion and Control conditions.

Diagram B: Comparison of Total Fat Oxidation and Ra FFA between Control and Lipid Infusion conditions.

Romijn et al. 1995
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<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Type 2 diabetes (n=8)</th>
<th>Obese controls (n=8)</th>
<th>Lean controls (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48±4</td>
<td>37±2</td>
<td>43±3</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>102±7</td>
<td>108±6</td>
<td>82±4&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>32±2</td>
<td>32±1</td>
<td>25±1&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>LBM (kg)</td>
<td>65±4</td>
<td>71±4</td>
<td>61±2&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>33±2</td>
<td>32±1</td>
<td>23±1&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Time since diagnosis (years)</td>
<td>4±1</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>$\dot{V}O_2_{max}$ leg (ml kg⁻¹ min⁻¹)</td>
<td>26±2</td>
<td>31±2&lt;sup&gt;c,e&lt;/sup&gt;</td>
<td>42±1&lt;sup&gt;a,e&lt;/sup&gt;</td>
</tr>
<tr>
<td>$\dot{V}O_2_{max}$ arm (ml kg⁻¹ min⁻¹)</td>
<td>22±2</td>
<td>22±1&lt;sup&gt;e&lt;/sup&gt;</td>
<td>31±2&lt;sup&gt;a,e&lt;/sup&gt;</td>
</tr>
<tr>
<td>Arm vs leg ratio ($\dot{V}O_2_{max}$)</td>
<td>0.82±0.06&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.71±0.03</td>
<td>0.73±0.05</td>
</tr>
<tr>
<td>IPAQ energy expenditure in a normal week (kJ)</td>
<td>641±148</td>
<td>541±103</td>
<td>732±147</td>
</tr>
</tbody>
</table>

Data are means±SE

Larsen et al. 2009
Larsen et al. 2009

Kang et al. 2007
Conclusion (1)

- During exercise with a limited muscle mass fat oxidation did not decrease during high intensity exercise, thus contrasting whole body exercise, where fat oxidation peaks at low to moderate intensities.

- No evidence for VLDL-TG utilization during one leg kicking at different exercise intensities!

- The difference between models implies that oxygen and/or substrate availability play a key role in the high intensity induced attenuation of fat oxidation during whole body exercise.
Conclusion (2)

- Lower absolute fat oxidation in arm versus leg muscle and lower exercise intensity eliciting maximal fat oxidation.

- Maintained maximal fat oxidation capacity during exercise in type II diabetes patients compared to matched obese and lean controls.

- Maintained maximal fat oxidation capacity during exercise in age-matched obese compared to age-matched lean controls.
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