Chapter 18
Electron Transport System
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Electron Transport System

We Are Here
Coupling Oxidation with Phosphorylation
Standard Reduction Potentials

Solution of 1M X and 1M X\(^-\)

1M H\(^+\) in equilibrium with 1 atm H\(_2\) gas
\[ \Delta G^\circ' = -nF \Delta E_0' \]

\[ F = 23000 \text{ cal/V mole} \]

### Table 18.1 Standard reduction potentials of some reactions

<table>
<thead>
<tr>
<th>Oxidant</th>
<th>Reductant</th>
<th>( n )</th>
<th>( E'_0 ) (V)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinate + CO(_2)</td>
<td>( \alpha )-Ketoglutarate</td>
<td>2</td>
<td>-0.67</td>
</tr>
<tr>
<td>Acetate</td>
<td>Acetaldehyde</td>
<td>2</td>
<td>-0.60</td>
</tr>
<tr>
<td>Ferredoxin (oxidized)</td>
<td>Ferredoxin (reduced)</td>
<td>1</td>
<td>-0.43</td>
</tr>
<tr>
<td>2 H(^+)</td>
<td>H(_2)</td>
<td>2</td>
<td>-0.42</td>
</tr>
<tr>
<td>NAD(^+)</td>
<td>NADH + H(^+)</td>
<td>2</td>
<td>-0.32</td>
</tr>
<tr>
<td>NADP(^+)</td>
<td>NADPH + H(^+)</td>
<td>2</td>
<td>-0.32</td>
</tr>
<tr>
<td>Lipoate (oxidized)</td>
<td>Lipoate (reduced)</td>
<td>2</td>
<td>-0.29</td>
</tr>
<tr>
<td>Glutathione (oxidized)</td>
<td>Glutathione (reduced)</td>
<td>2</td>
<td>-0.23</td>
</tr>
<tr>
<td>FAD</td>
<td>FADH(_2)</td>
<td>2</td>
<td>-0.22</td>
</tr>
<tr>
<td>Acetaldehyde</td>
<td>Ethanol</td>
<td>2</td>
<td>-0.20</td>
</tr>
<tr>
<td>Pyruvate</td>
<td>Lactate</td>
<td>2</td>
<td>-0.19</td>
</tr>
<tr>
<td>Fumarate</td>
<td>Succinate</td>
<td>2</td>
<td>+0.03</td>
</tr>
<tr>
<td>Cytochrome b (+3)</td>
<td>Cytochrome b (+2)</td>
<td>1</td>
<td>+0.07</td>
</tr>
<tr>
<td>Dehydroascorbate</td>
<td>Ascorbate</td>
<td>2</td>
<td>+0.08</td>
</tr>
<tr>
<td>Ubiquinone (oxidized)</td>
<td>Ubiquinone (reduced)</td>
<td>2</td>
<td>+0.10</td>
</tr>
<tr>
<td>Cytochrome c (+3)</td>
<td>Cytochrome c (+2)</td>
<td>1</td>
<td>+0.22</td>
</tr>
<tr>
<td>Fe (+3)</td>
<td>Fe (+2)</td>
<td>1</td>
<td>+0.77</td>
</tr>
<tr>
<td>( \frac{1}{2} \text{O}_2 + 2 \text{H}^+ )</td>
<td>H(_2)O</td>
<td>2</td>
<td>+0.82</td>
</tr>
</tbody>
</table>

Note: \( E'_0 \) is the standard oxidation–reduction potential (pH 7, 25°C) and \( n \) is the number of electrons transferred. \( E'_0 \) refers to the partial reaction written as

\[ \text{Oxidant} + e^- \rightarrow \text{reductant} \]
But what transfers electrons?
The cytochrome system

David Keilin, 1925!!! Discovered cytochromes, Ochoa, 1945 linked Oxidation with Phosphorylation
From KEGG database
Chemiosmotic Theory: Peter Mitchell 1961

Protons are pumped across this membrane as electrons flow through the respiratory chain.

More usefully, for ejection of one H+ from the matrix:

\[ \Delta G (kJ \text{ or kcal/mol}) = RT \ln ([H^+]_{\text{cytosol}}/[H^+]_{\text{matrix}}) + F \Delta \Psi \]

\[ = 2.3 \, RT \, (\text{pH}_{\text{matrix}} - \text{pH}_{\text{cytosol}}) + F \Delta \Psi \]

\[ \text{Pmf (volts)} = \Delta \Psi + \left( \frac{RT \ln 10}{F} \right) \Delta \text{pH} \]
Chemiosmotic Theory: Charged Mitochondria are easily labeled
Chemiosmotic Theory Principles:
The membrane as capacitor

- \( V = \frac{q}{c} \)

\( q \) = charge (in Coulombs, \( 1e^- = 1.6 \times 10^{-19} \text{ C/electron charge} \))

\( V \) = voltage in Volts

\( F \), Farads

\( c \) = capacitance
Chemiosmotic Theory: The membrane as capacitor

- Q: How many H^+’s need to be pumped by an ATP’ase across a spherical bacterium (r=1.0 µm) to get a transmembrane voltage of −60. mV? Bilayer capacitance = 1.0 x 10^{-6} F/cm^2. Area of a sphere=4π r^2 Volume of a sphere=4/3π r^3
The proof: Racker and Stoeckennius, 1974
J Biol Chem--

Also a Frankenstein experiment! Knox and Tsong
### Table 18.2 Components of the mitochondrial electron-transport chain

<table>
<thead>
<tr>
<th>Enzyme complex</th>
<th>Mass (kd)</th>
<th>Subunits</th>
<th>Prosthetic group</th>
<th>Oxidant or Reductant</th>
<th>Matrix side</th>
<th>Membrane core</th>
<th>Cytoplasmic side</th>
</tr>
</thead>
<tbody>
<tr>
<td>NADH-Q oxidoreductase</td>
<td>&gt;900</td>
<td>46</td>
<td>FMN</td>
<td>NADH</td>
<td>Q</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Fe-S</td>
<td></td>
<td>Succinate</td>
<td></td>
<td>Cytochrome c</td>
</tr>
<tr>
<td>Succinate-Q reductase</td>
<td>140</td>
<td>4</td>
<td>FAD</td>
<td>Succinate</td>
<td>Q</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Fe-S</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q-cytochrome c oxidoreductase</td>
<td>250</td>
<td>11</td>
<td>Heme b&lt;sub&gt;H&lt;/sub&gt;</td>
<td></td>
<td>Q</td>
<td></td>
<td>Cytochrome c</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Heme b&lt;sub&gt;L&lt;/sub&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Heme c&lt;sub&gt;1&lt;/sub&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Fe-S</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cytochrome c oxidase</td>
<td>160</td>
<td>13</td>
<td>Heme a</td>
<td></td>
<td></td>
<td></td>
<td>Cytochrome c</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Heme a&lt;sub&gt;2&lt;/sub&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cu&lt;sub&gt;A&lt;/sub&gt; and Cu&lt;sub&gt;B&lt;/sub&gt;</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Table 18-2  
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Overview of ETS-see Conceptual Insight

Overview animation

http://bcs.whfreeman.com/stryer/pages/bcs-main.asp?s=00010&n=99000&i=99010.01&v=category&o=&ns=0&uid=110354&rau=110354
BACK to the DETAILS! ETS supramolecular organization??


http://www.jbc.org/cgi/content/full/282/1/1

MINIREVIEW: OXPHOS Supercomplexes of Mitochondria

FIGURE 3. Model of the supramolecular structure of the OXPHOS system: single complexes co-exist with supramolecular assemblies. Complex I (red) can associate with complex III, (blue). Complex III, can associate with one or two copies of complex IV (purple). The largest assemblies include complex I, dimeric complex III, and one or several copies of complex IV. Yellow circles, ubiquinol, which either freely diffuses within the inner mitochondrial membrane or might form part of the I+III2 supercomplex. For simplicity, complex II was omitted from the figure because it is not known to form part of OXPHOS supercomplexes. Furthermore, cytochrome c, alternative oxidoreductases, and the ATP synthase complex are omitted from the figure. Modified from Bianchi et al. (27).

FIGURE 2. Structure of mitochondrial OXPHOS supercomplexes as revealed by single particle electron microscopy. A, top view projection map of the I+III2 supercomplex of Arabidopsis (25); B, side view map of the III2+IV2 supercomplex of yeast; C, side view map of dimeric ATP synthase of Polytomella (39). In the schemes protein complexes or large protein domains are indicated by colors. A: complex III2, blue; complex I, green (light green, peripheral arm; medium green, membrane arm; dark green, carbonic anhydrase domain). B: complex III2, blue; complex IV, purple. C: F0 parts, red; F1 parts and central stalks, yellow; peripheral stalks, orange.
Figure 18-7
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Flavin mononucleotide (oxidized) (FMN)

Semiquinone intermediate

Flavin mononucleotide (reduced) (FMNH₂)
NADH Dehydrogenase or NADH-Q oxidoreductase
The b-c complex or Q-cytochrome c oxidoreductase
C-type cytochromes
1. Two molecules of cytochrome c sequentially transfer electrons to reduce Cu_B and heme α_3.

2. Reduced Cu_B and Fe in heme α_3 bind O_2, which forms a peroxide bridge.

2 H_2O

3. The addition of two more electrons and two more protons cleaves the peroxide bridge.

4. The addition of two more protons leads to the release of water.

Figure 18-14
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Overall

Figure 18-16
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# The Danger of using Oxygen as an oxidizing agent!

**Table 18.3 Pathological and other conditions that may entail free-radical injury**

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atherogenesis</td>
</tr>
<tr>
<td>Emphysema; bronchitis</td>
</tr>
<tr>
<td>Parkinson disease</td>
</tr>
<tr>
<td>Duchenne muscular dystrophy</td>
</tr>
<tr>
<td>Cervical cancer</td>
</tr>
<tr>
<td>Alcoholic liver disease</td>
</tr>
<tr>
<td>Diabetes</td>
</tr>
<tr>
<td>Acute renal failure</td>
</tr>
<tr>
<td>Down syndrome</td>
</tr>
<tr>
<td>Retrolental fibroplasia (conversion of the retina into a fibrous mass in premature infants)</td>
</tr>
<tr>
<td>Cerebrovascular disorders</td>
</tr>
<tr>
<td>Ischemia; reperfusion injury</td>
</tr>
</tbody>
</table>

**ROS**

<table>
<thead>
<tr>
<th>Reactive oxygen species</th>
<th>Unpaired electrons</th>
</tr>
</thead>
<tbody>
<tr>
<td>O₂</td>
<td>−</td>
</tr>
<tr>
<td>O₂⁻</td>
<td>−</td>
</tr>
<tr>
<td>O₂⁻²</td>
<td>−</td>
</tr>
<tr>
<td>OH⁻</td>
<td>−</td>
</tr>
</tbody>
</table>

- Oxygen
- Superoxide anion
- Peroxide
- Hydroxyl radical
- Hydroxyl ion
Oxygen Radical Damage
Oxygen Radical Scavengers: small molecules

Vitamin C

Vitamin e

glutathione

Beta carotene
Oxygen Radical Scavengers: Enzymes

- Superoxide dismutase

\[
O_2^- + O_2^- \xrightarrow{\text{SOD}} O_2 + H_2O_2
\]

(+2 H\(^+\))

- Catalase: finishes the job

\[
2H_2O_2 \rightarrow 2H_2O + O_2
\]

- Glutathione peroxidase
The Danger of using Oxygen as an oxidizing agent!

SOD takes off the Edge

\[ \text{M}_{\text{ox}} \xrightarrow{O_2^-} \text{M}_{\text{red}} \xrightarrow{O_2} \text{M}_{\text{ox}} \]

\[ \text{M}_{\text{red}} \xleftarrow{H_2O_2} \text{M}_{\text{ox}} \xleftarrow{2H^+} \text{M}_{\text{red}} \]
The Final Payoff: the ATPase
Boyer’s Nobel Prize-winning model
Probing Nature's Nanoscale Machines with Microscale Probes

Kazuhiko Kinosita
Waseda University
Tokyo, Japan

"Mechanically driven ATP synthesis by F1-ATPase"
F1-ATPase Attached to an Actin Filament

The F1 portion of ATP synthase consists of six stator subunits (green and blue) surrounding a central rotor driveshaft (orange). Here a fluorescent actin filament (red) is attached to the driveshaft via a streptavidin linkage (purple).
How Did they Stick the ATPase to the Glass Slide in the 1st Place?

- The F1 complex from a thermophilic bacterium was expressed in E coli with 10 histidines linked to the N terminus of each beta subunit.
- Glass plate was coated with horseradish peroxidase conjugated with Ni$^{2+}$ nitrilotriacetic acid.
How did they stick the actin to it?

- Through site directed mutagenesis of the gamma subunit they replaced Ser107 with Cys193 (the only Cys in the wild type gamma)
- They biotinylated the Cys and the fluorescent actin
- The two were attached through streptavidin which has 4 binding sites for biotin
What did they see?

Rotation of the actin filaments
Propeller-type rotation was key to showing this is a true rotary motor

http://www.k2.phys.waseda.ac.jp/F1movies/F1Prop.htm
Smallest Known Rotary Motor

• Central rotor, the gamma subunit, is 1 nm in radius and the barrel in which it rotates, made up of the beta and alpha subunits, is 5nm in radius

• In the presence of 2 mM ATP, the F1 ATPase rotated the actin filament CCW 100+ revolutions and produced a constant torque of about 40 pN nm
"Coupling of rotation and catalysis in F1-ATPase revealed by single-molecule imaging and manipulation"

http://www.k2.phys.waseda.ac.jp/Publications.html
Mechanically Driven ATP Synthesis by F1-ATPase

- Attached a magnetic bead to the gamma subunit of the F1 ATPase
- Rotated the bead using electrical magnets to apply torque in medium containing ADP & P_i
Mechanically Driven ATP Synthesis by F1-ATPase

- CW rotation leads to the production of ATP
- ATP was detected using luciferin-luciferase, which emits a photon of light when it captures and hydrolyzes ATP and by simply turning off the electromagnetic force and observing a change in direction of rotation which requires the hydrolysis of ATP
Figure 18-31

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Glycerol-3-Phosphate shuttle

“Loses” part of an NADH

\[
\begin{align*}
\text{NADH} + \text{H}^+ & \rightarrow \text{NAD}^+ \\
\text{Dihydroxyacetone phosphate} & \rightarrow \text{Glycerol 3-phosphate}
\end{align*}
\]
Malate/Aspartate shuttle-in heart and liver

“Saves” an ATP
Getting it out! The ATP translocase

Figure 18-37
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Figure 18-39
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Problem

For, summing up synthesis of \(~P \text{ bonds}\) via oxidative phosphorylation, assume:

2.5 \(~P \text{ bonds}\) synthesized during oxidation of \(\text{NADH}\) produced via \textbf{Pyruvate Dehydrogenase & Krebs Cycle} (10 \(H^+\) pumped; 4 \(H^+\) used up per ATP).

1.5 \(~P \text{ bonds}\) synthesized per \(\text{NADH}\) produced in the cytosol in \textbf{Glycolysis} (electrons transferred via \(\text{FAD}\) to coenzyme \(Q\)).

1.5 \(~P \text{ bonds}\) synthesized during oxidation of \(\text{FADH}_2\) produced in \textbf{Krebs Cycle} (Succinate Dehydrogenase – electrons transferred to coenzyme \(Q\)).
## All Quantities Per Glucose

<table>
<thead>
<tr>
<th>Pathway</th>
<th>NADH produced</th>
<th>FADH$_2$ produced (QH$_2$)</th>
<th>~P bonds ATP or GTP direct</th>
<th>~P bonds 1.5 or 2.5 per NADH in oxphos</th>
<th>~P bonds 1.5 per FADH$_2$ in oxphos</th>
<th>Total ~P bonds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycolysis Pathway</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pyruvate Dehydrogenase</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Krebs Cycle</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sum of Pathways</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Table 18.4 ATP yield from the complete oxidation of glucose

<table>
<thead>
<tr>
<th>Reaction sequence</th>
<th>ATP yield per glucose molecule</th>
</tr>
</thead>
</table>
| **Glycolysis: Conversion of glucose into pyruvate**
(in the cytoplasm) |                               |
| Phosphorylation of glucose | −1                           |
| Phosphorylation of fructose 6-phosphate | −1                           |
| Dephosphorylation of 2 molecules of 1,3-BPG | +2                           |
| Dephosphorylation of 2 molecules of phosphoenolpyruvate | +2                           |
| 2 molecules of NADH are formed in the oxidation of 2 molecules of glyceraldehyde 3-phosphate |       |
| **Conversion of pyruvate into acetyl CoA**
(inside mitochondria) |                               |
| 2 molecules of NADH are formed |                               |
| **Citric acid cycle (inside mitochondria)** | +2                           |
| 2 molecules of guanosine triphosphate are formed from | +2 |
| 2 molecules of succinyl CoA |                               |
| 6 molecules of NADH are formed in the oxidation of 2 molecules each of isocitrate, α-ketoglutarate, and malate | +2 |
| 2 molecules of FADH$_2$ are formed in the oxidation of 2 molecules of succinate | +2 |
| **Oxidative phosphorylation (inside mitochondria)** | +3                           |
| 2 molecules of NADH formed in glycolysis; each yields 1.5 molecules of ATP (assuming transport of NADH by the glycerol 3-phosphate shuttle) | +3 |
| 2 molecules of NADH formed in the oxidative decarboxylation of pyruvate; each yields 2.5 molecules of ATP | +5 |
| 2 molecules of FADH$_2$ formed in the citric acid cycle; each yields 1.5 molecules of ATP | +3 |
| 6 molecules of NADH formed in the citric acid cycle; each yields 2.5 molecules of ATP | +15 |

**NET YIELD PER MOLECULE OF GLUCOSE**

+ 30


Note: The current value of 30 molecules of ATP per molecule of glucose supersedes the earlier one of 36 molecules of ATP. The stoichiometries of proton pumping, ATP synthesis, and metabolite transport should be regarded as estimates. About two more molecules of ATP are formed per molecule of glucose oxidized when the malate-aspartate shuttle rather than the glycerol 3-phosphate shuttle is used.

Table 18-4
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An **oxygen electrode** may be used to record \([O_2]\) in a closed vessel. Electron transfer, e.g., \(\text{NADH} \rightarrow \text{O}_2\), is monitored by the rate of \(\text{O}_2\) disappearance.

Above is represented an \(\text{O}_2\) electrode recording while mitochondria respire in the presence of \(\text{P}_i\) and an \(e^-\) donor (succinate or a substrate of a reaction to generate \(\text{NADH}\)).

The dependence of respiration rate on availability of \(\text{ADP}\), the ATP Synthase substrate, is called **respiratory control**.
**Chemiosmotic explanation of respiratory control:**
Electron transfer is obligatorily coupled to H\(^+\) ejection from the matrix. Whether this coupled reaction is spontaneous depends on pH and electrical gradients.

<table>
<thead>
<tr>
<th>Reaction</th>
<th>(\Delta G)</th>
</tr>
</thead>
<tbody>
<tr>
<td>e(^-) transfer (NADH (\rightarrow) O(_2))</td>
<td>negative value*</td>
</tr>
<tr>
<td>H(^+) ejection from matrix</td>
<td>positive; depends on H(^+) gradient**</td>
</tr>
<tr>
<td>e(^-) transfer with H(^+) ejection</td>
<td>algebraic sum of above</td>
</tr>
</tbody>
</table>

\(\Delta G^o = -nF\Delta E^o = -218 \text{ kJ/mol for } 2e^- \text{ NADH} \rightarrow \text{O}_2\).

**For ejection of 1 H\(^+\) from the matrix:**
\[\Delta G = RT \ln \left(\frac{[H^+]_{cytosol}}{[H^+]_{matrix}}\right) + F\Delta\Psi\]
\[\Delta G = 2.3 RT \left(pH_{matrix} - pH_{cytosol}\right) + F\Delta\Psi\]
Respiratory control ratio is the ratio of slopes after and before ADP addition \((b/a)\).

P/O ratio is the moles of ADP divided by the moles of O consumed (based on \(c\)) while phosphorylating the ADP.
With **no ADP**, H$^+$ cannot flow through F$_o$. ΔpH & ΔΨ are maximal. As respiration/H$^+$ pumping proceed, **ΔG for H$^+$ ejection increases**, approaching that for e$^-$ transfer.

When the coupled reaction is non-spontaneous, **respiration stops**. This is referred to as a **static head**.

In fact there is usually a low rate of respiration in the absence of ADP, attributed to H$^+$ leaks.
When ADP is added, H\(^+\) enters the matrix via F\(_o\), as ATP is synthesized. This reduces \(\Delta p\text{H} \& \Delta \Psi\).

**\(\Delta G\) of H\(^+\) ejection decreases.**

The coupled reaction of electron transfer with H\(^+\) ejection becomes spontaneous.

Respiration resumes or is stimulated.
Inhibitors of electron transport:
Tools and Poisons

Figure 18-43
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Uncouplers: Mitchell’s revenge!!
Uncouplers: Mitchell’s revenge!!

Dinitrophenol and obesity: An early twentieth-century regulatory dilemma

Eric Colman

Abstract

In the early 1930s, the industrial chemical dinitrophenol found widespread use due principally to the work of Maurice Tainter, a clinical pharmacologist. Unfortunately, the compound’s therapeutic index was razed, and thousands of people suffered irreversible harm that mainstream physicians did not foresee. The risks outweighed its benefits and led to its abandonment. Food, Drug, and Cosmetic Act in 1938 before federal regulators had medicine men from selling dinitrophenol to Americans lured by the promise of a safe way to melt one’s fat away.

Mitochondrial uncoupling as a target for drug development for the treatment of obesity.

Harper JA, Dickinson K, Brand MD.

MRC Dunn Human Nutrition Unit, Hills Road, Cambridge CB2 2XY, UK.

Mitochondrial proton cycling is responsible for a significant proportion of basal or standard metabolic rate, so further uncoupling of mitochondria may be a good way to increase energy expenditure and represents a good pharmacological target for the treatment of obesity. Uncoupling by 2,4-dinitrophenol has been used in this way in the past with notable success, and some of the effects of thyroid hormone treatment to induce weight loss may also be due to uncoupling. Diet can alter the pattern of phospholipid fatty acid groups in the mitochondrial membrane, and this may be a route to uncoupling in vivo. Energy expenditure can be increased by stimulating the activity of uncoupling protein 1 (UCP1) in brown adipocytes either directly or through beta 3-adrenergic agonists. UCP2 in a number of tissues, UCP3 in skeletal muscle and the adenine nucleotide translocase have also been proposed as possible drug targets. Specific uncoupling of muscle or brown adipocyte mitochondria remains an attractive target for the development of antiobesity drugs.

PMID: 12119996 [PubMed - indexed for MEDLINE]
Thermogenesis for dieting:
enough biochemistry to be dangerous???

http://www.sea-thin.com/

fucoxanthin
Thermogenesis for dieting???

Upregulation of UCP-1 normally found mostly in Brown Fat?

Effect of Medium-chain Triacylglycerols on Anti-obesity Effect of Fucoxanthin

Hayato Maeda1*, Masashi Hosokawa1, Tokutake Sashima2, Katsura Funayama3 and Kazuo Miyashita1

1 Faculty of Fisheries Sciences, Hokkaido University (Hakodate, Hokkaido 041-8611, JAPAN)
2 Creative Research Institute, Hokkaido University (Hakodate, Hokkaido 041-8611, JAPAN)
3 Riken Vitamin Co., (Tokyo 174-0065, JAPAN)

Abstract: Dietary effects of medium-chain triacylglycerols (MCT) and fucoxanthin (Fc) on abdominal fat weight were determined using KK-Ay obese mouse. Experimental diet contained MCT (0.9%), Fc (0.1%), or MCT (0.9%) + Fc (0.1%). The abdominal fat weight of mice fed with Fc was significantly lower than that of mice fed with MCT. Uncoupling protein 1 (UCP1), a key molecule for metabolic thermogenesis, was clearly expressed in the white adipose tissue (WAT) of mice fed Fc, but little expression in that of the mice fed MCT. The anti-obesity effect of Fc was increased by mixing Fc with MCT. This increase would be due to the increase in the absorption rate of Fc by MCT.

Keywords: fucoxanthin, obesity, medium-chain triacylglycerol

5 mg fucoxanthin- note that 0.1% would be part of a 5000 mg diet - ummm.....