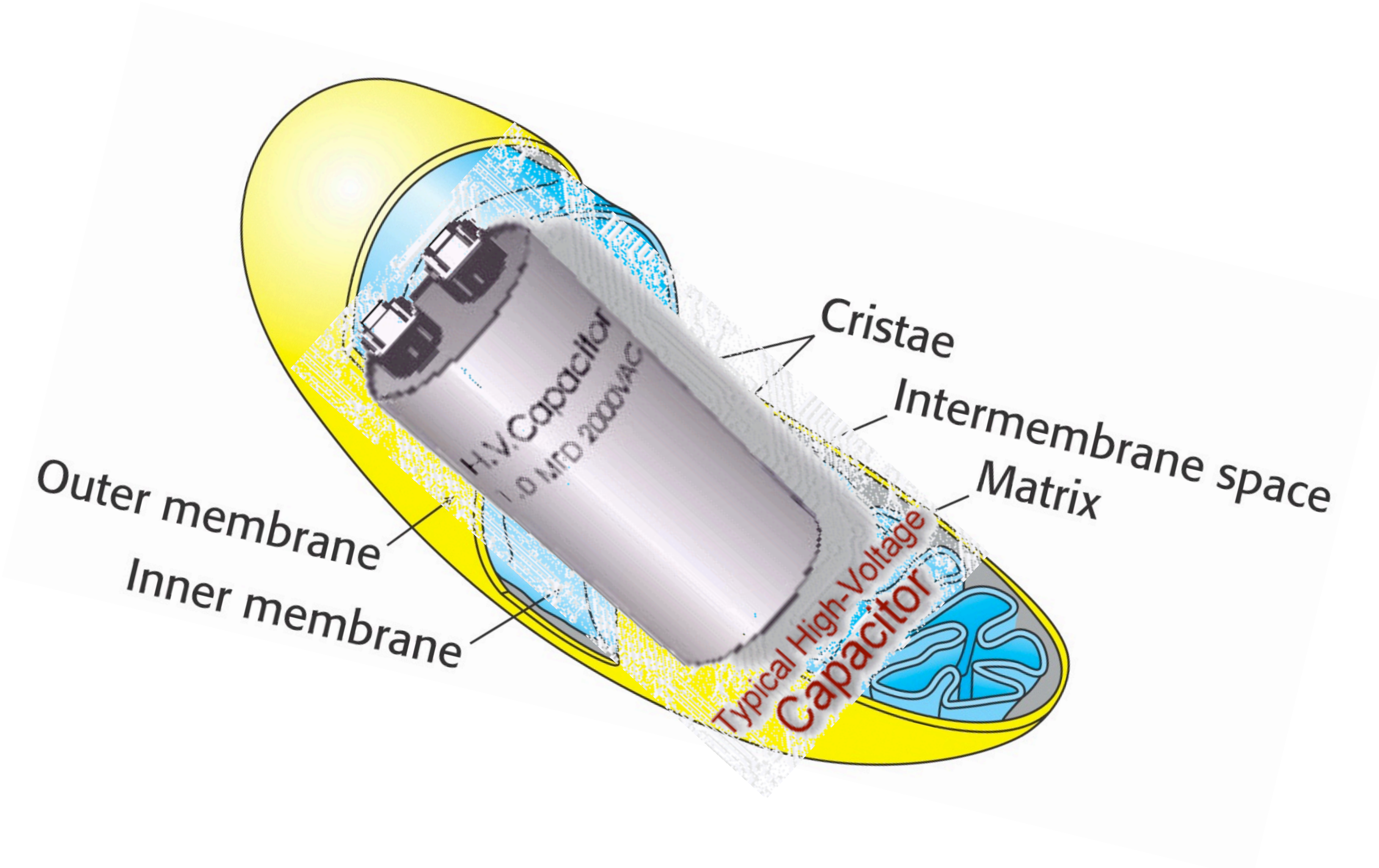
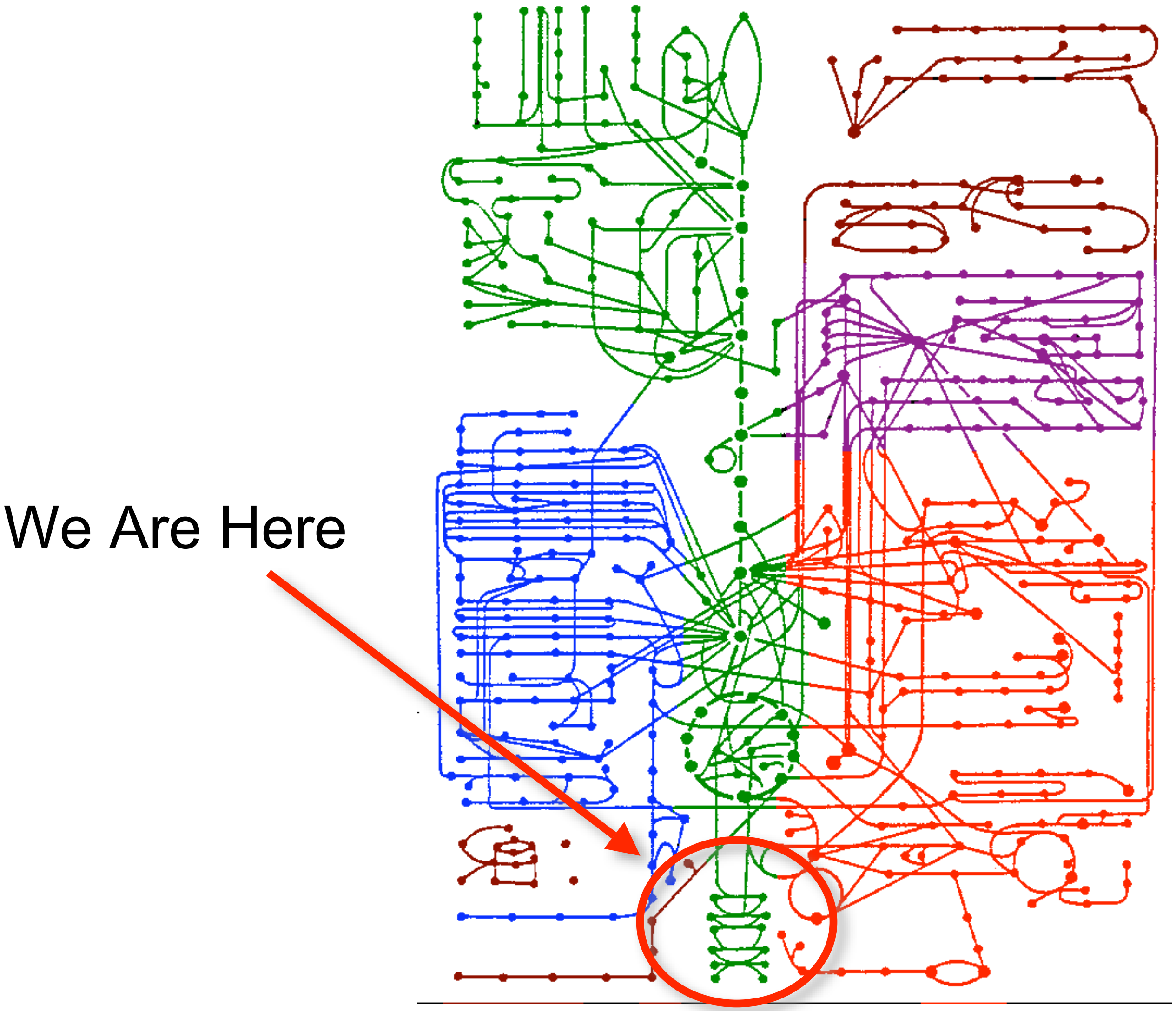
Chapter 18
Electron Transport System

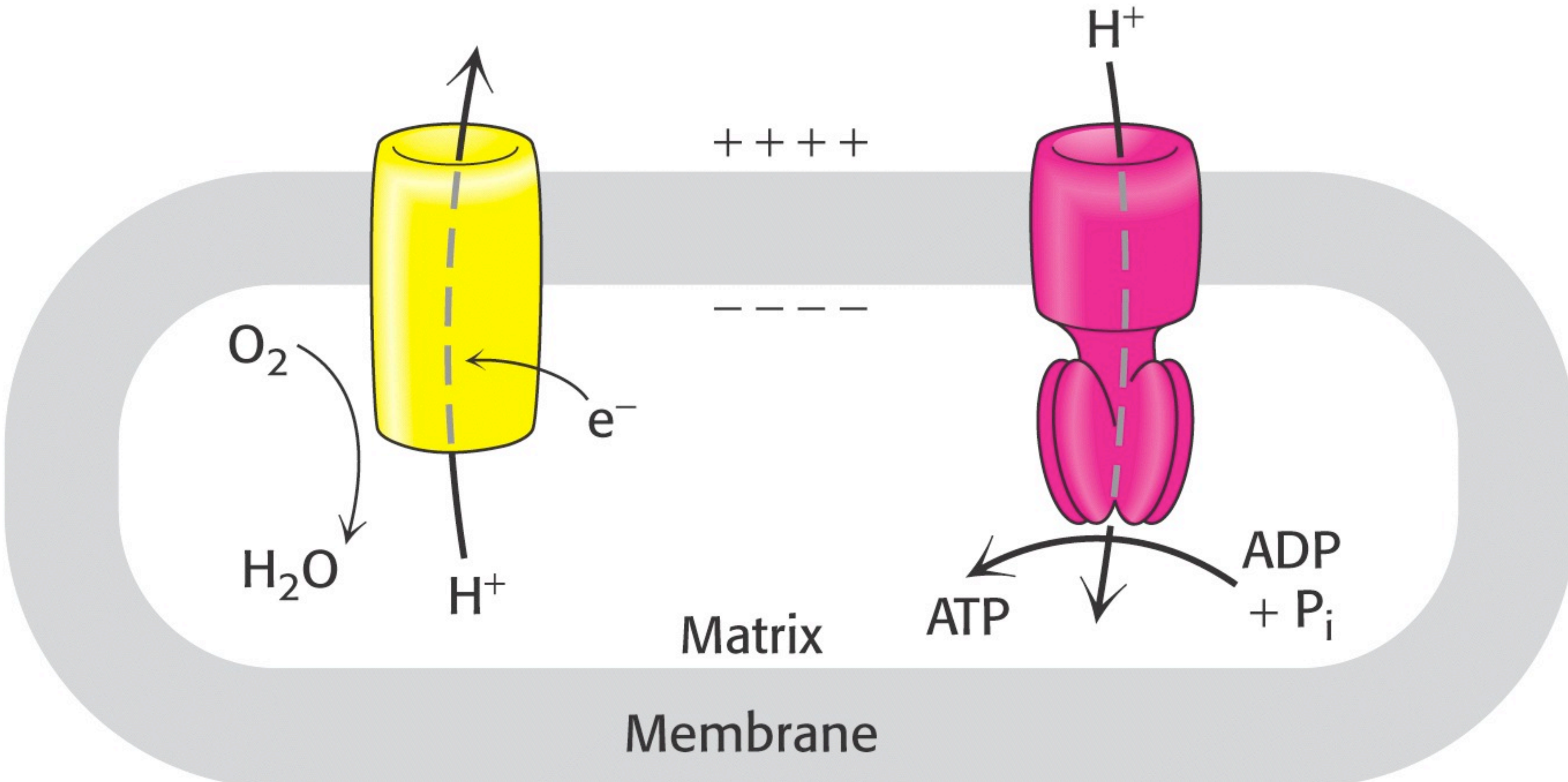


Chapter 18

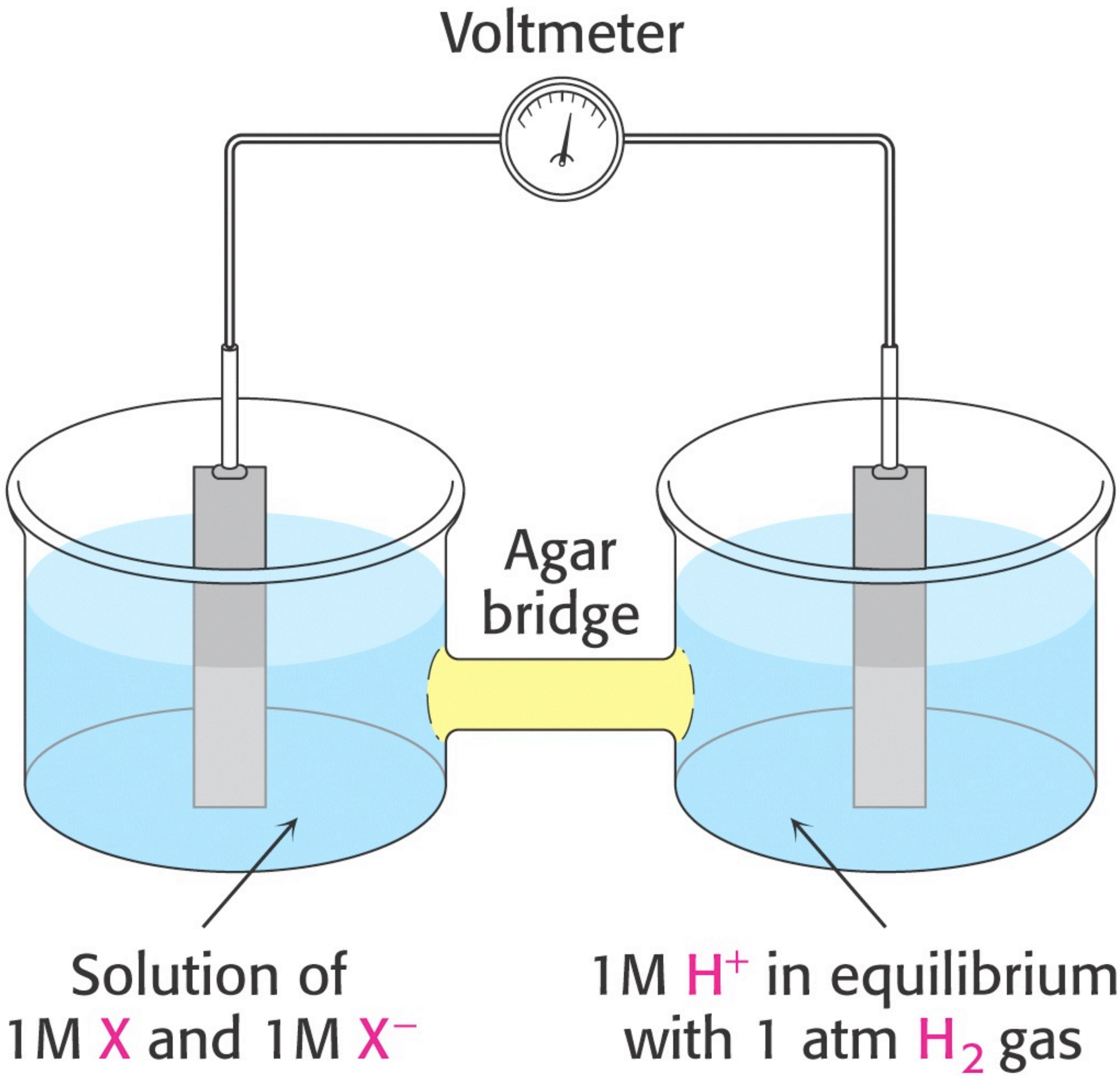
Electron Transport System



Coupling Oxidation with Phosphorylation



Standard Reduction Potentials



$\Delta G^{o\prime} = - nF\Delta E_{o}{}^{\prime}$

acceptor- donor= $\Delta E_{o}{}'$

F = 23000 cał /V mole

TABLE 18.1 Standard reduction potentials of some reactions

Oxidant	Reductant	*n*	E'_0 (V)
Succinate + CO_2	α-Ketoglutarate	2	−0.67
Acetate	Acetaldehyde	2	−0.60
Ferredoxin (oxidized)	Ferredoxin (reduced)	1	−0.43
2 H^+	H_2	2	−0.42
NAD^+	NADH + H^+	2	−0.32
$NADP^+$	NADPH + H^+	2	−0.32
Lipoate (oxidized)	Lipoate (reduced)	2	−0.29
Glutathione (oxidized)	Glutathione (reduced)	2	−0.23
FAD	$FADH_2$	2	−0.22
Acetaldehyde	Ethanol	2	−0.20
Pyruvate	Lactate	2	−0.19
Fumarate	Succinate	2	+0.03
Cytochrome *b* (+3)	Cytochrome *b* (+2)	1	+0.07
Dehydroascorbate	Ascorbate	2	+0.08
Ubiquinone (oxidized)	Ubiquinone (reduced)	2	+0.10
Cytochrome *c* (+3)	Cytochrome *c* (+2)	1	+0.22
Fe (+3)	Fe (+2)	1	+0.77
½ O_2 + 2 H^+	H_2O	2	+0.82

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

Oxidant + e^- → reductant

Table 18-1
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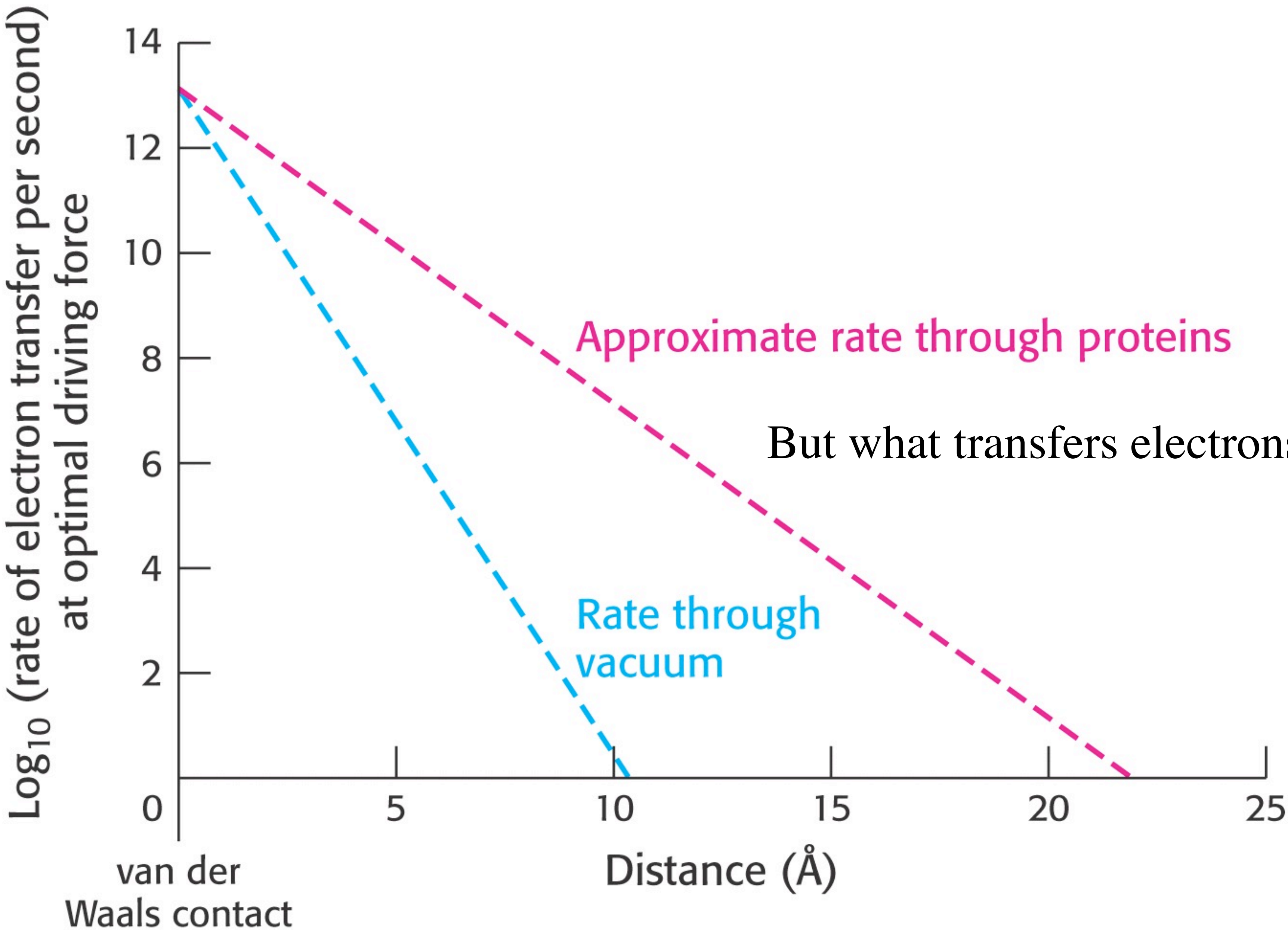
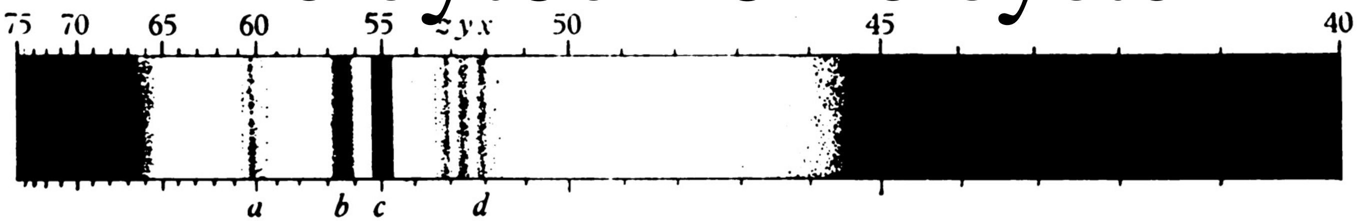
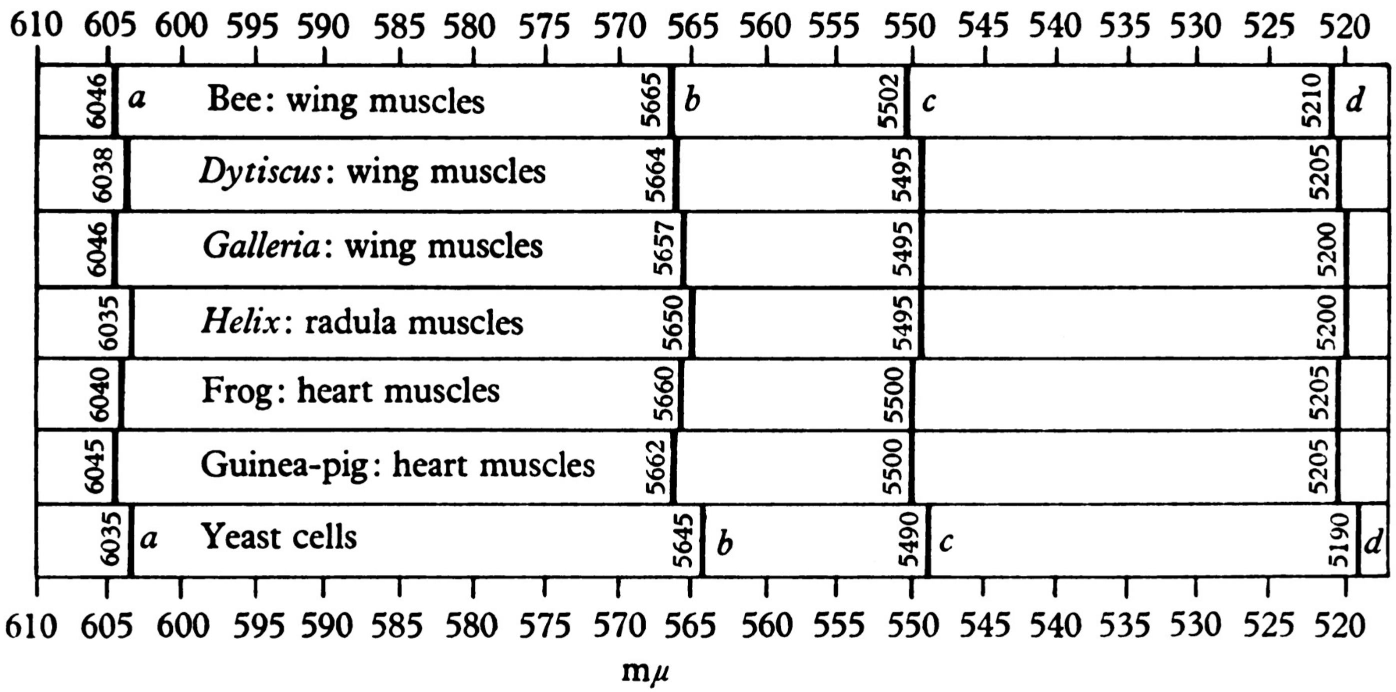


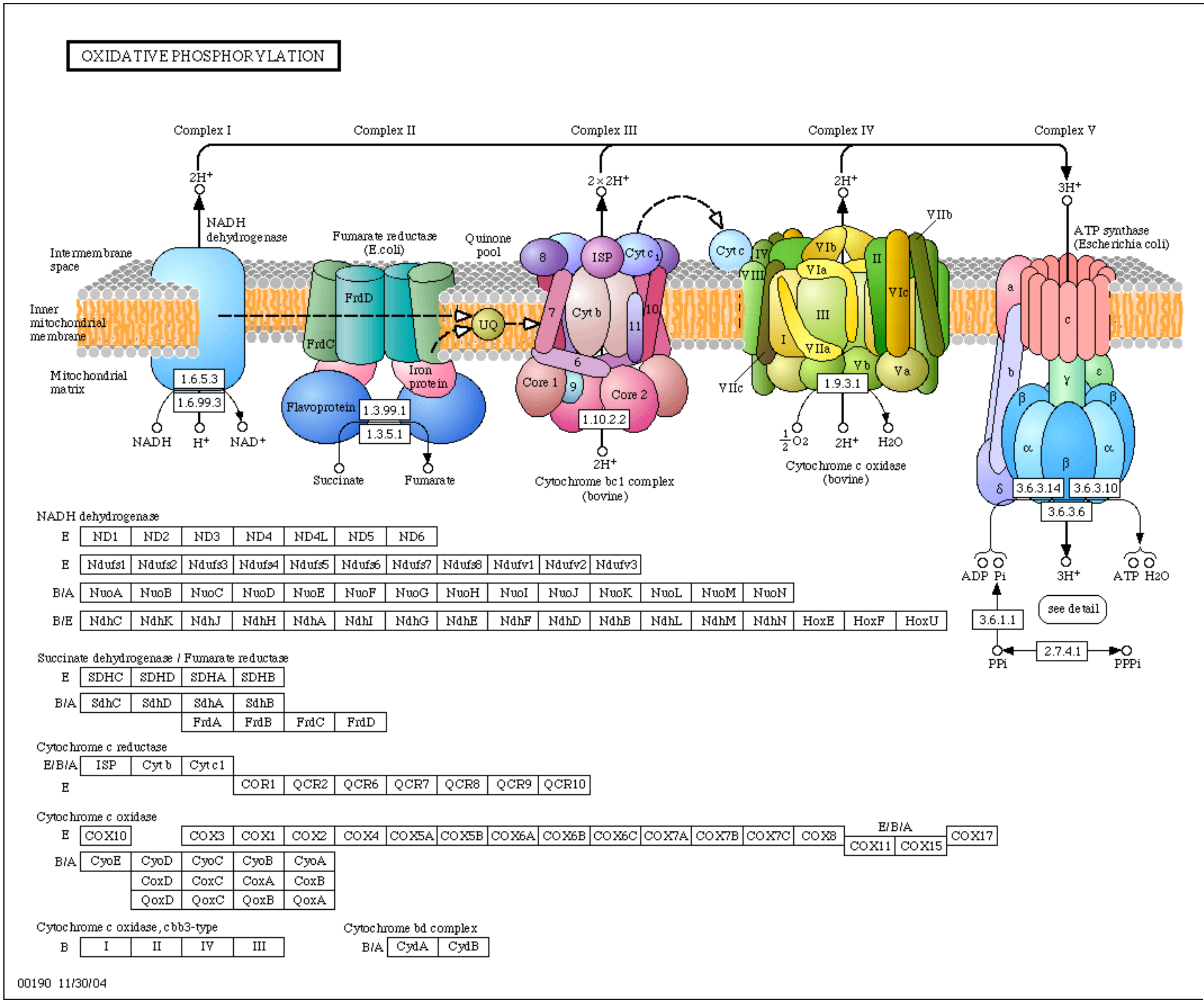
But what transfers electrons?

The cytochrome system





David Keilin, 1925!!! Discovered cytochromes, Ochoa, 1945 linked Oxidation with Phosphorylation



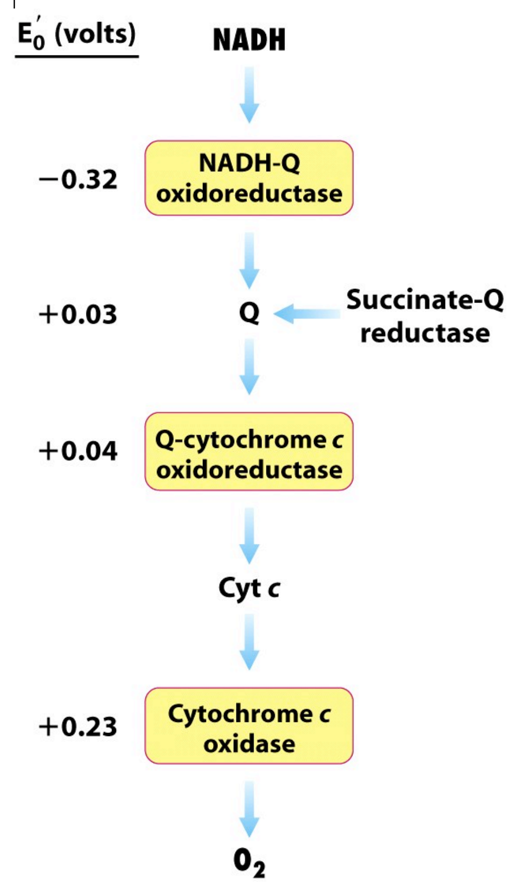


Figure 18-6
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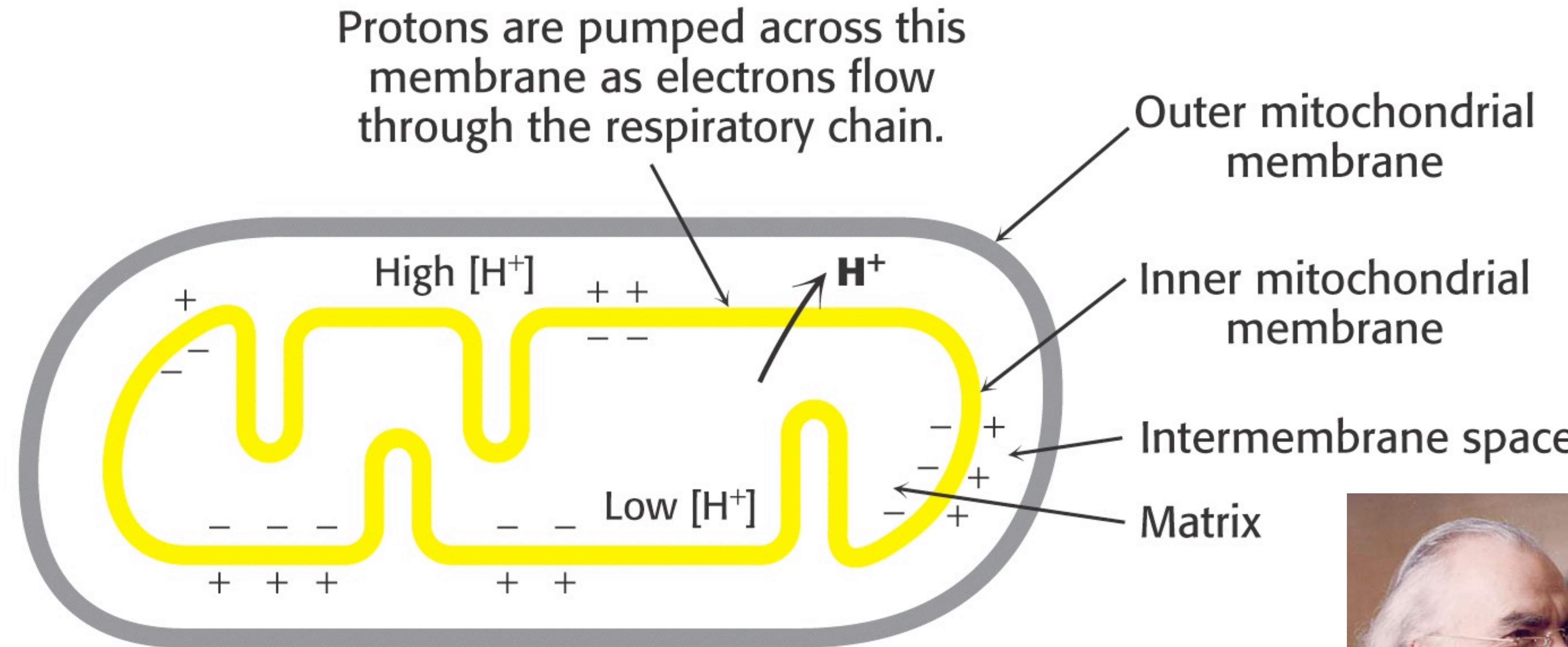
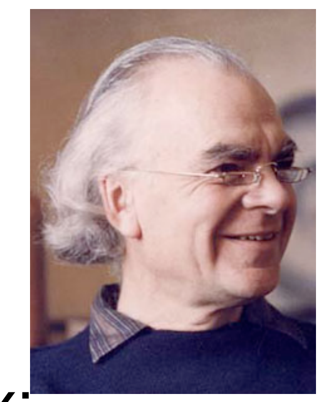
From KEGG database

Chemiosmotic Theory: Peter Mitchell 1961





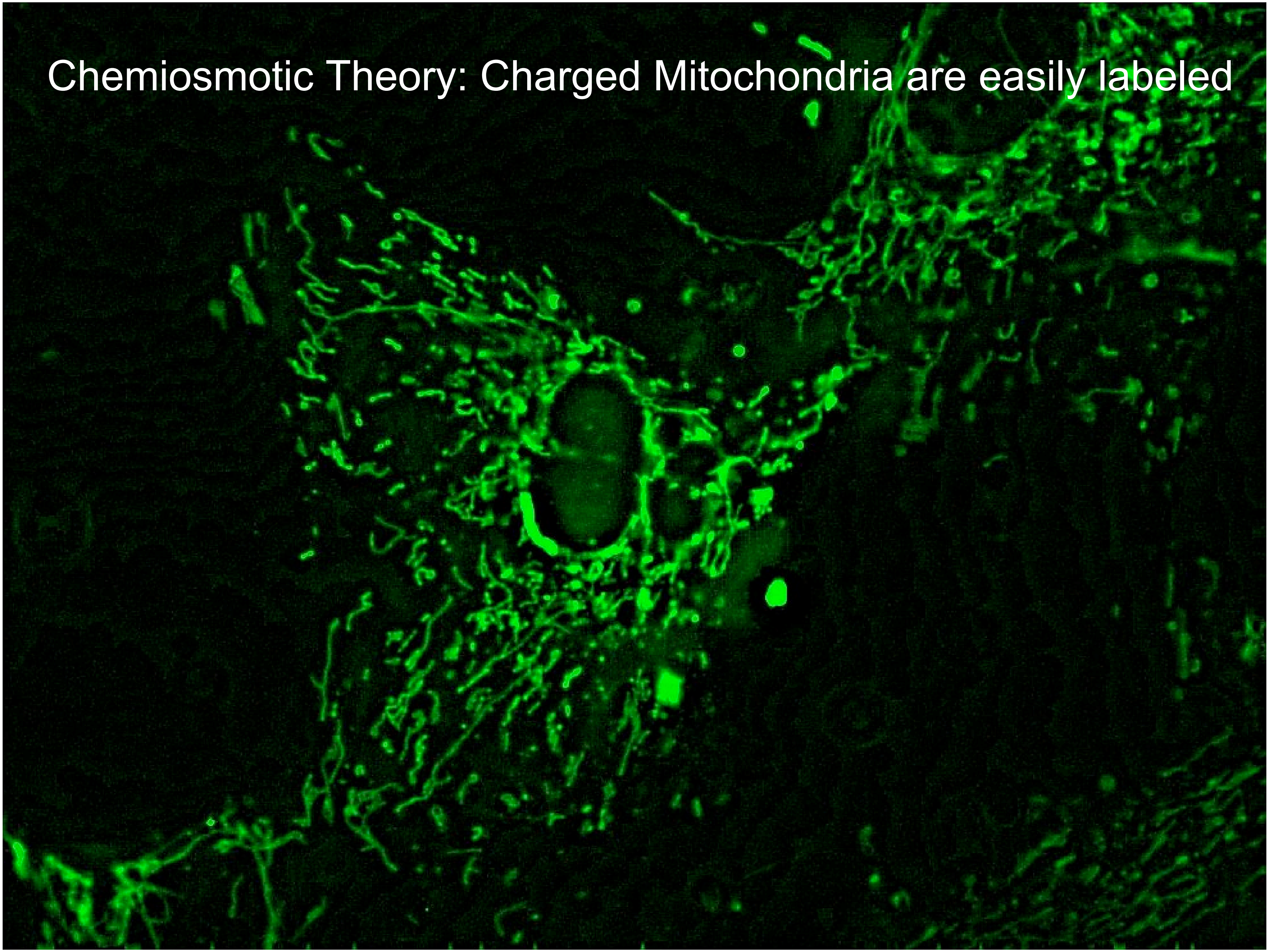
$$\text{Pmf (volts)} = \Delta\Psi + (RT \ln 10/F)\,\Delta pH$$

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

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

$$= 2.3\,RT\,(pH_{matrix} - pH_{cytosol}) + F\,\Delta\Psi$$

$$\text{Pmf (volts)} = \Delta\Psi + (RT \ln 10/F)\,\Delta pH$$


Chemiosmotic Theory: Charged Mitochondria are easily labeled

Chemiosmotic Theory Principles:
The membrane as capacitor

- $V = q/c$

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

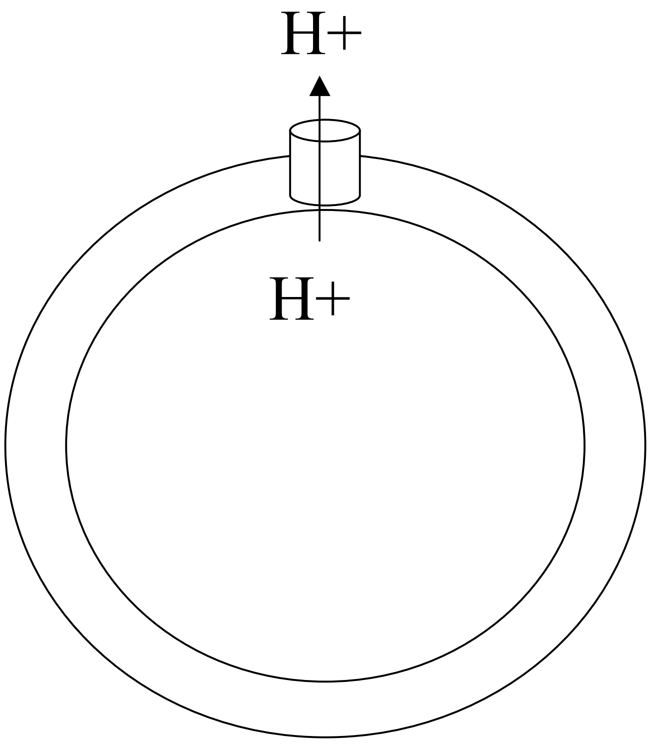
V=voltage in Volts

(F, Farads)

c= capacitance

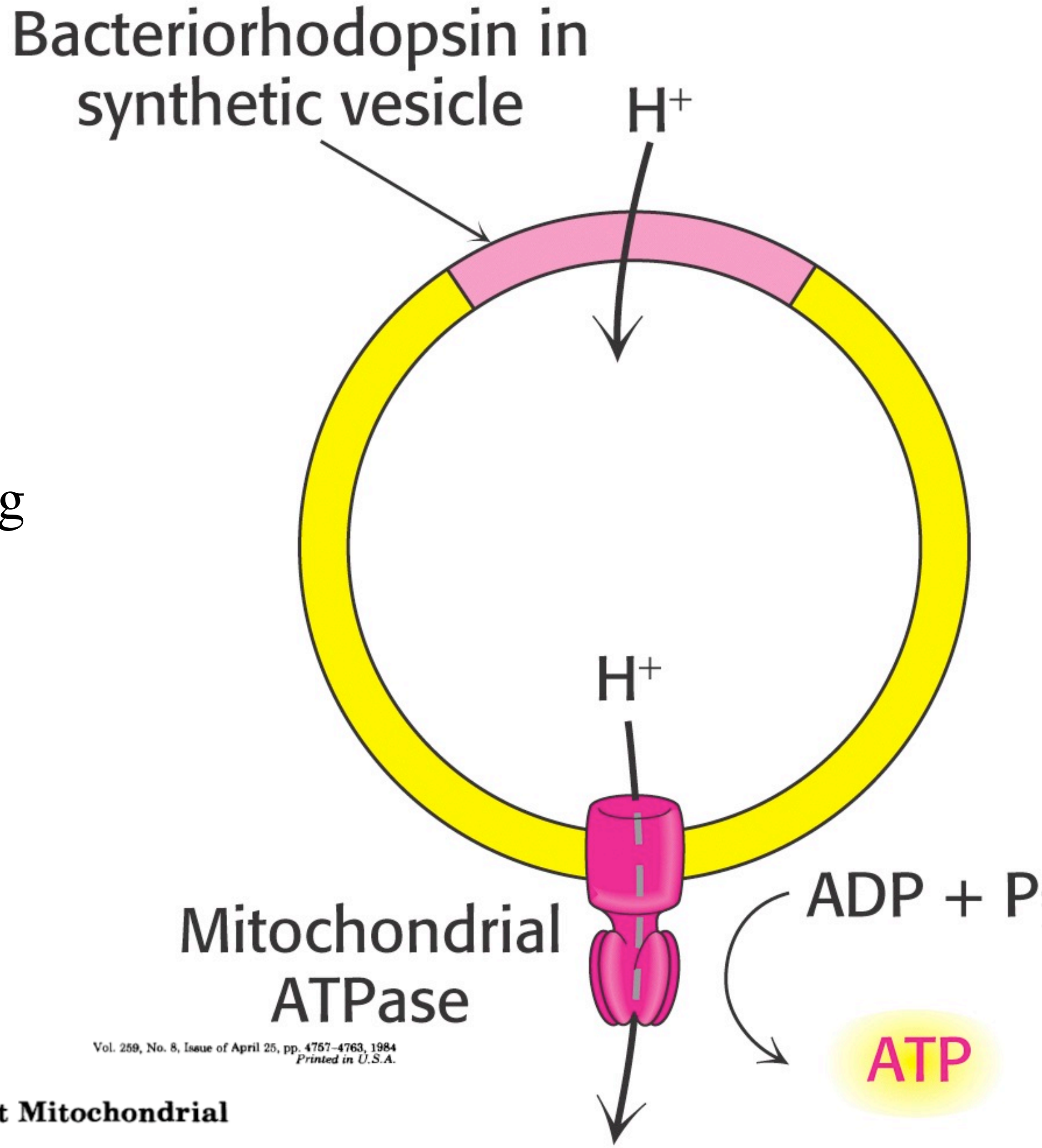
Chemiosmotic Theory:The membrane as capacitor

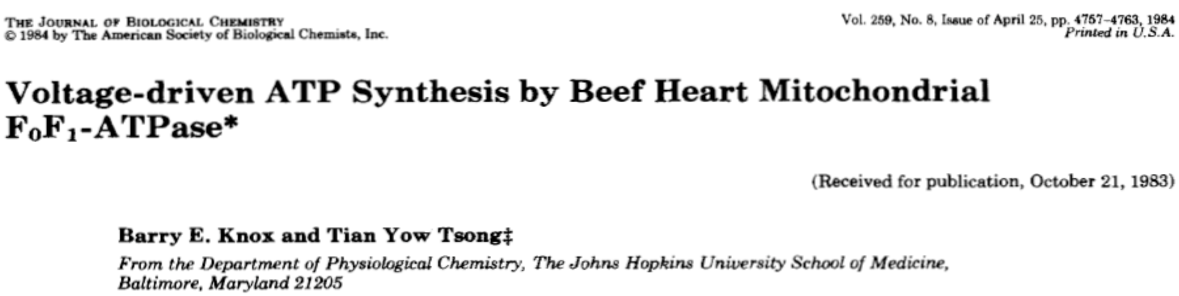
- Q: How many H^+'s need to be pumped by an ATP'ase across a spherical bacterium (r=1.0 μm) to get a transmembrane voltage of –60. mV? Bilayer capacitance = 1.0 x 10^{-6} F/cm^2. Area of a sphere=4π r^2 Volume of a sphere=4/3π r^3



The proof: Racker and Stoeckenius, 1974
J Biol Chem--

Also a Frankenstein experiment! Knox and Tsong

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Vol. 259, No. 8, Issue of April 25, pp. 4757-4763, 1984
Printed in U.S.A.

Voltage-driven ATP Synthesis by Beef Heart Mitochondrial F_0F_1-ATPase*

(Received for publication, October 21, 1983)

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From the Department of Physiological Chemistry, The Johns Hopkins University School of Medicine, Baltimore, Maryland 21205

BACK to the DETAILS!

TABLE 18.2 Components of the mitochondrial electron-transport chain

Enzyme complex	Mass (kd)	Subunits	Prosthetic group	OXIDANT OR REDUCTANT: Matrix side	Membrane core	Cytoplasmic side
NADH-Q oxidoreductase	>900	46	FMN Fe-S	NADH	Q	
Succinate-Q reductase	140	4	FAD Fe-S	Succinate	Q	
Q-cytochrome *c* oxidoreductase	250	11	Heme b_H Heme b_L Heme c_1 Fe-S		Q	Cytochrome *c*
Cytochrome *c* oxidase	160	13	Heme *a* Heme a_3 Cu_A and Cu_B			Cytochrome *c*

Sources: J. W. DePierre and L. Ernster. *Annu. Rev. Biochem.* 46(1977):215; Y. Hatefi. *Annu Rev. Biochem.* 54(1985);1015; and J. E. Walker. *Q. Rev. Biophys.* 25(1992):253.

Table 18-2
Biochemistry, Sixth Edition

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??

J. Biol. Chem., Vol. 282,
Issue 1, 1-4, January 5, 2007

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

MINIREVIEW: *OXPHOS Supercomplexes of Mitochondria*

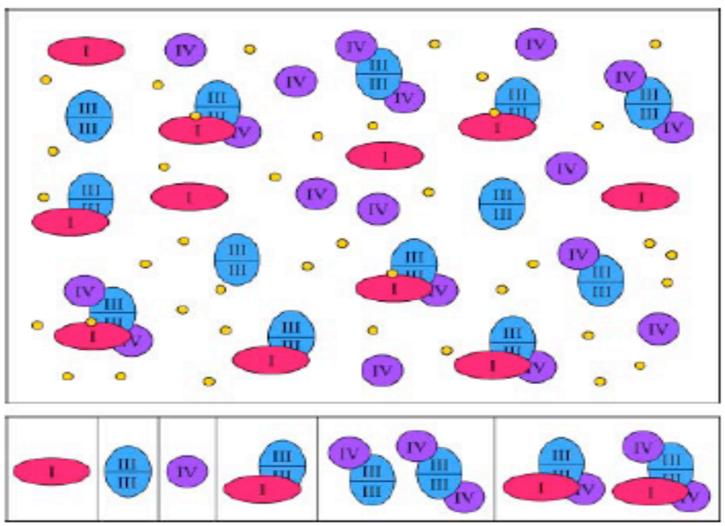

FIGURE 3. **Model of the supramolecular structure of the OXPHOS system: single complexes co-exist with supramolecular assemblies.** Complex I (*red*) can associate with complex III_2 (*blue*). Complex III_2 can associate with one or two copies of complex IV (*purple*). The largest assemblies include 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+III_2 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).

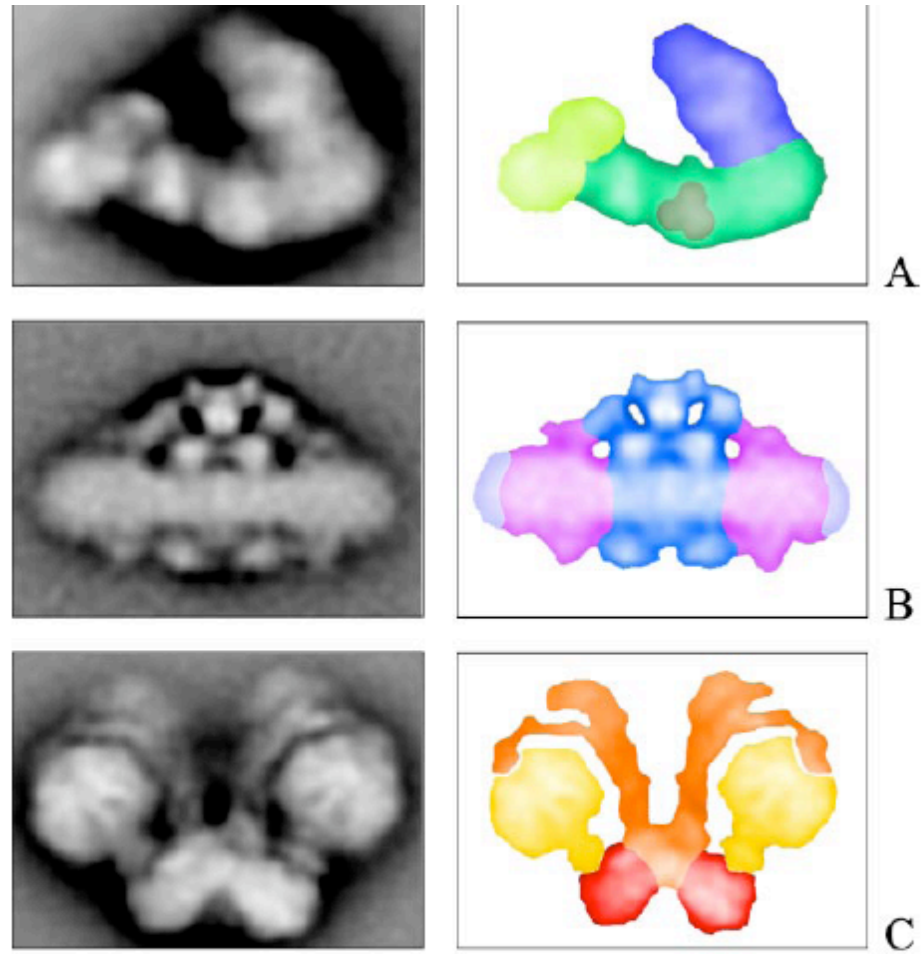

FIGURE 2. **Structure of mitochondrial OXPHOS supercomplexes as revealed by single particle electron microscopy.** *A*, top view projection map of the I+III_2 supercomplex of *Arabidopsis* (25); *B*, side view map of the III_2+IV_2 supercomplex of yeast[3]; *C*, side view map of dimeric ATP synthase of *Polytomella* (39). In the schemes protein complexes or large protein domains are indicated by *colors*. *A*: complex III_2, *blue*; complex I, *green* (*light green*, peripheral arm; *medium green*, membrane arm; *dark green*, carbonic anhydrase domain). *B*: complex III_2, *blue*; complex IV, *purple*. *C*: F_0 parts, *red*; F_1 parts and central stalks, *yellow*; peripheral stalks, *orange*.

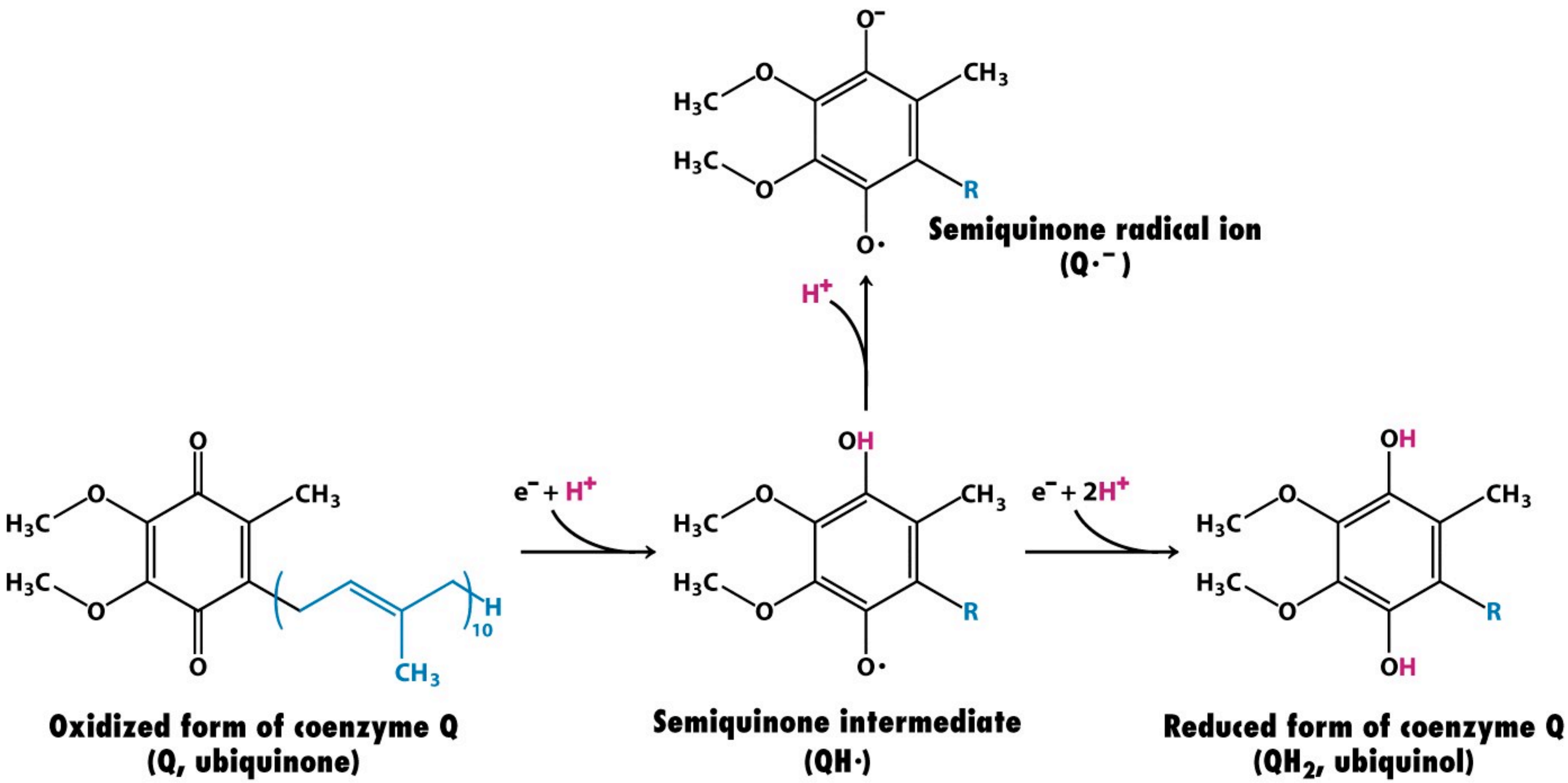

Figure 18-7
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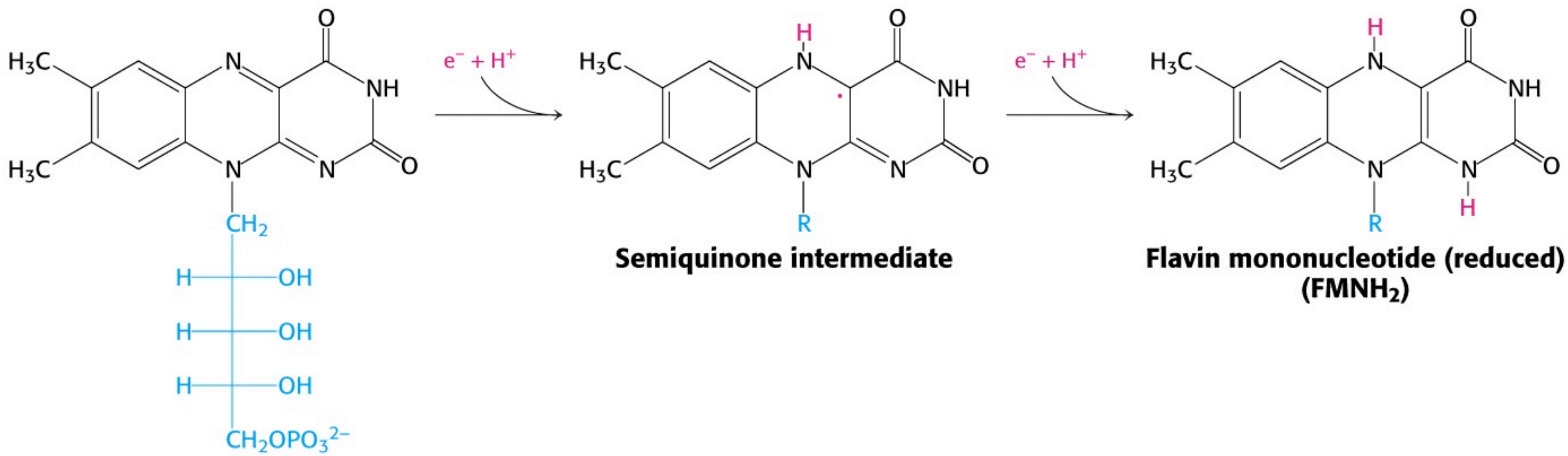
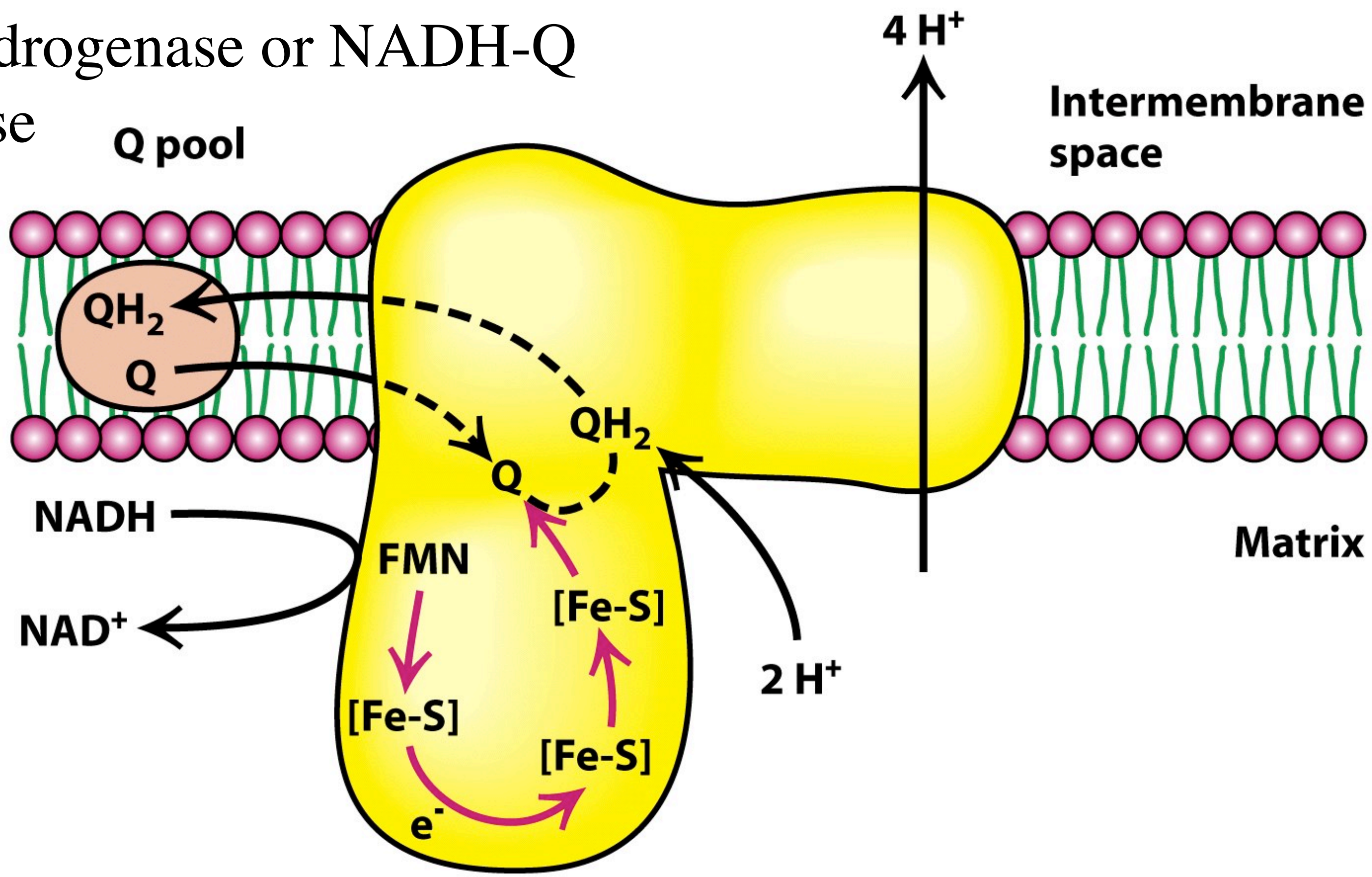


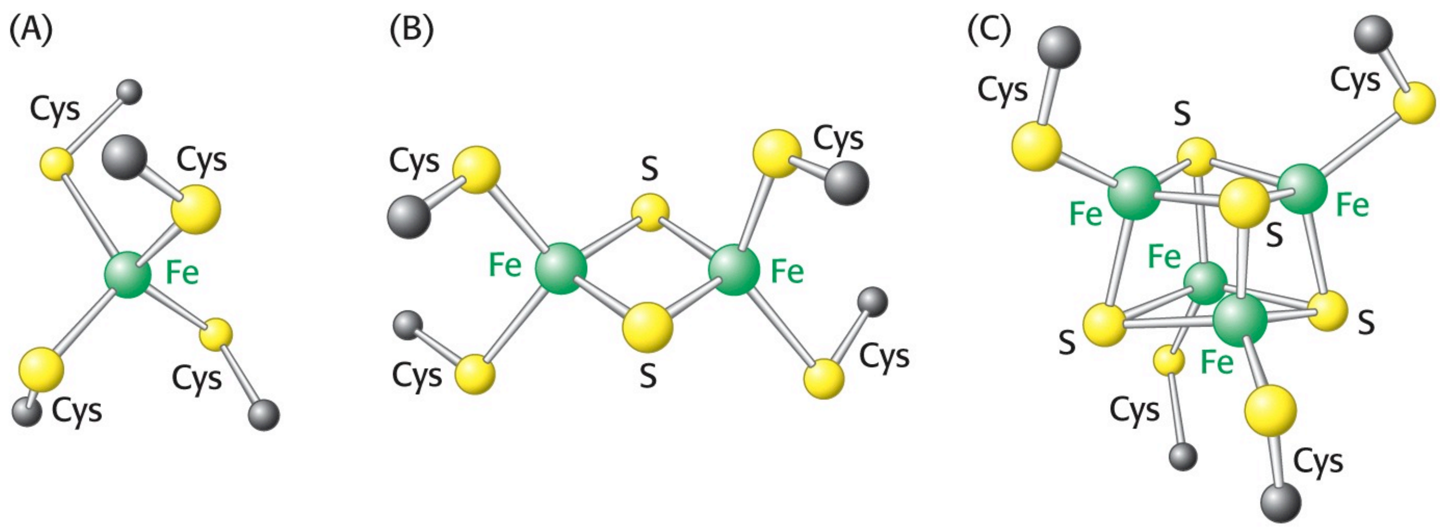
Flavin mononucleotide (oxidized)
(FMN)

Semiquinone intermediate

Flavin mononucleotide (reduced)
($FMNH_2$)

NADH Dehydrogenase or NADH-Q oxidoreductase





The b-c complex or
Q-cytochrome c oxidoreductase

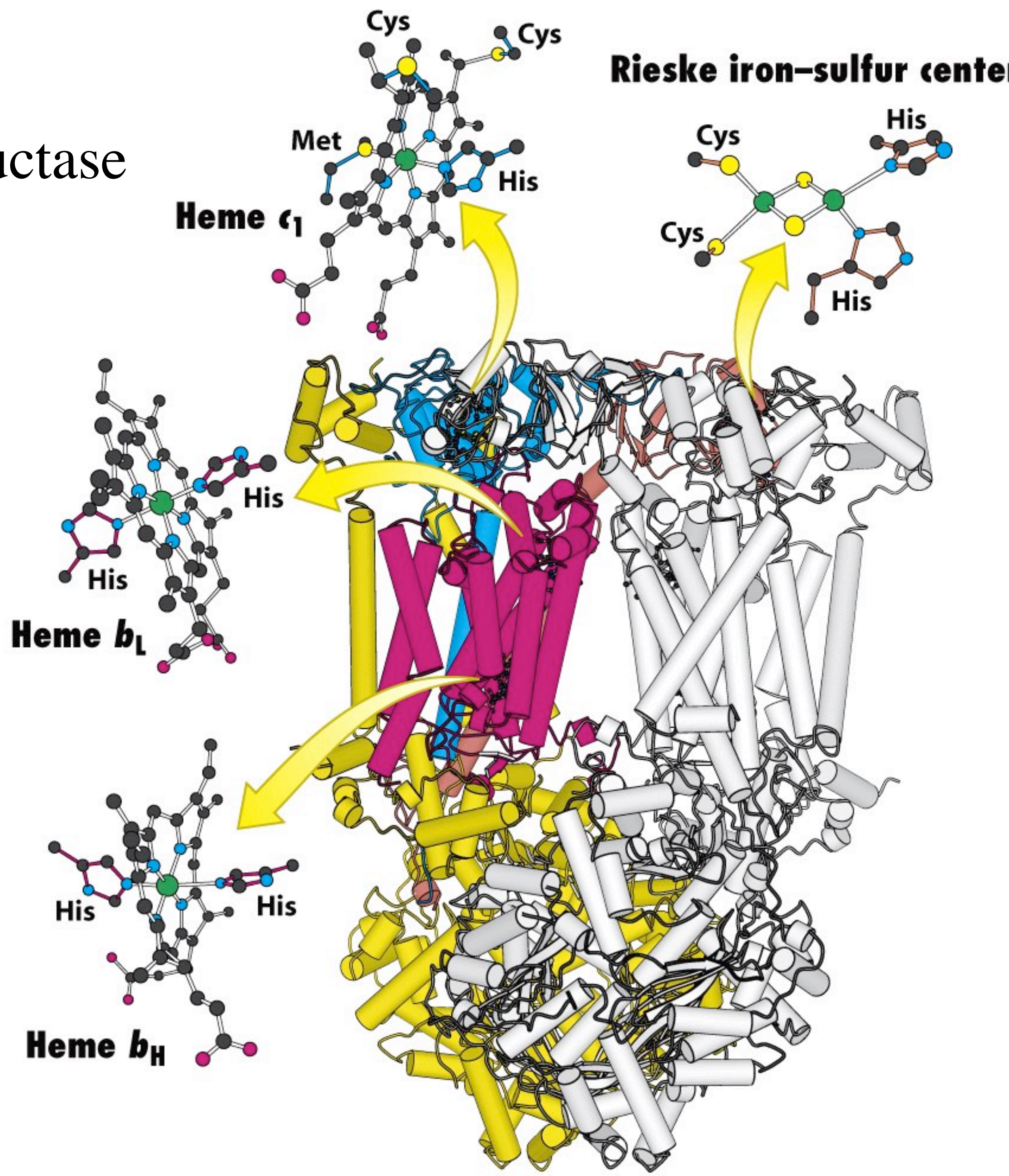


Figure 18-11
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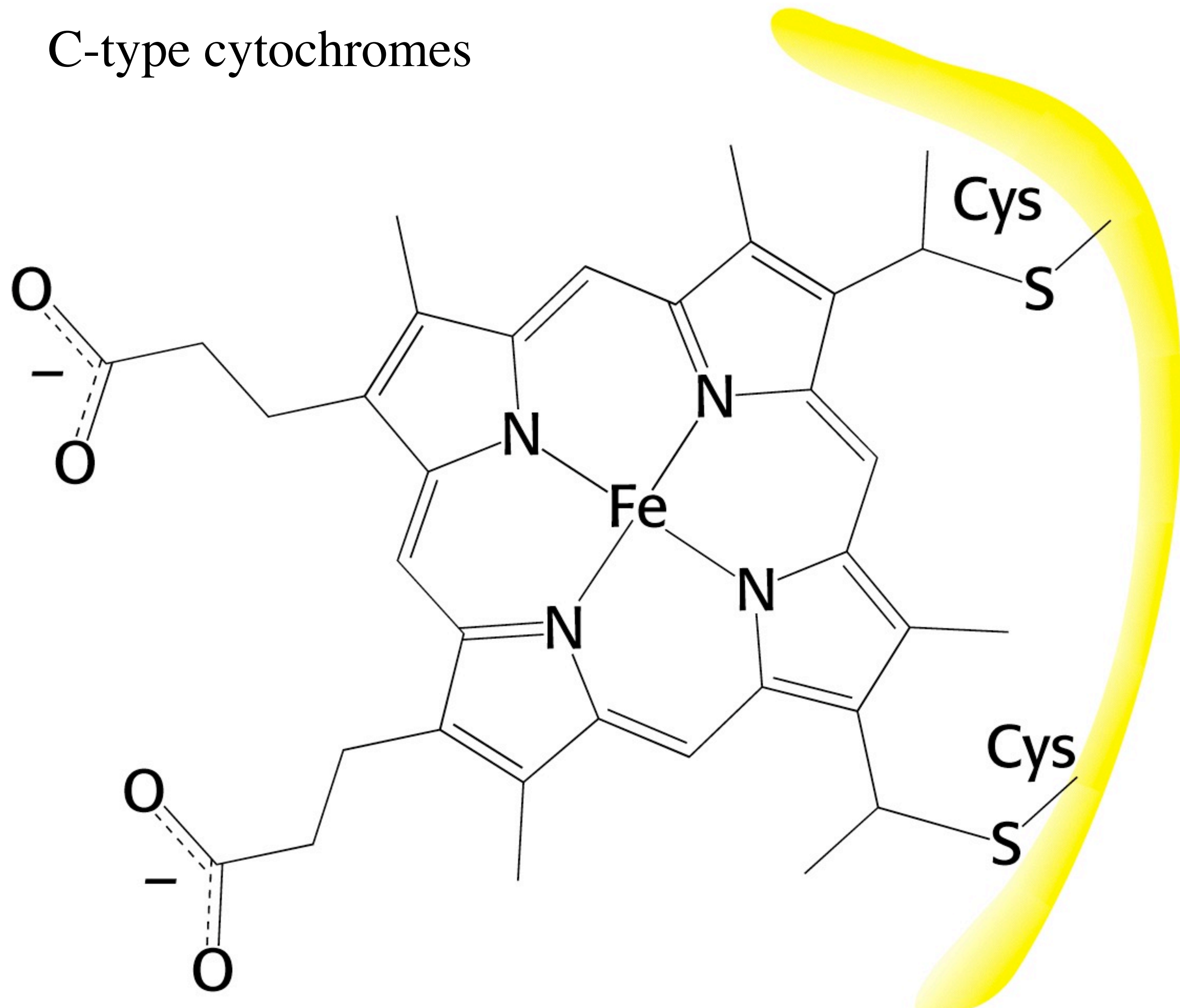
C-type cytochromes



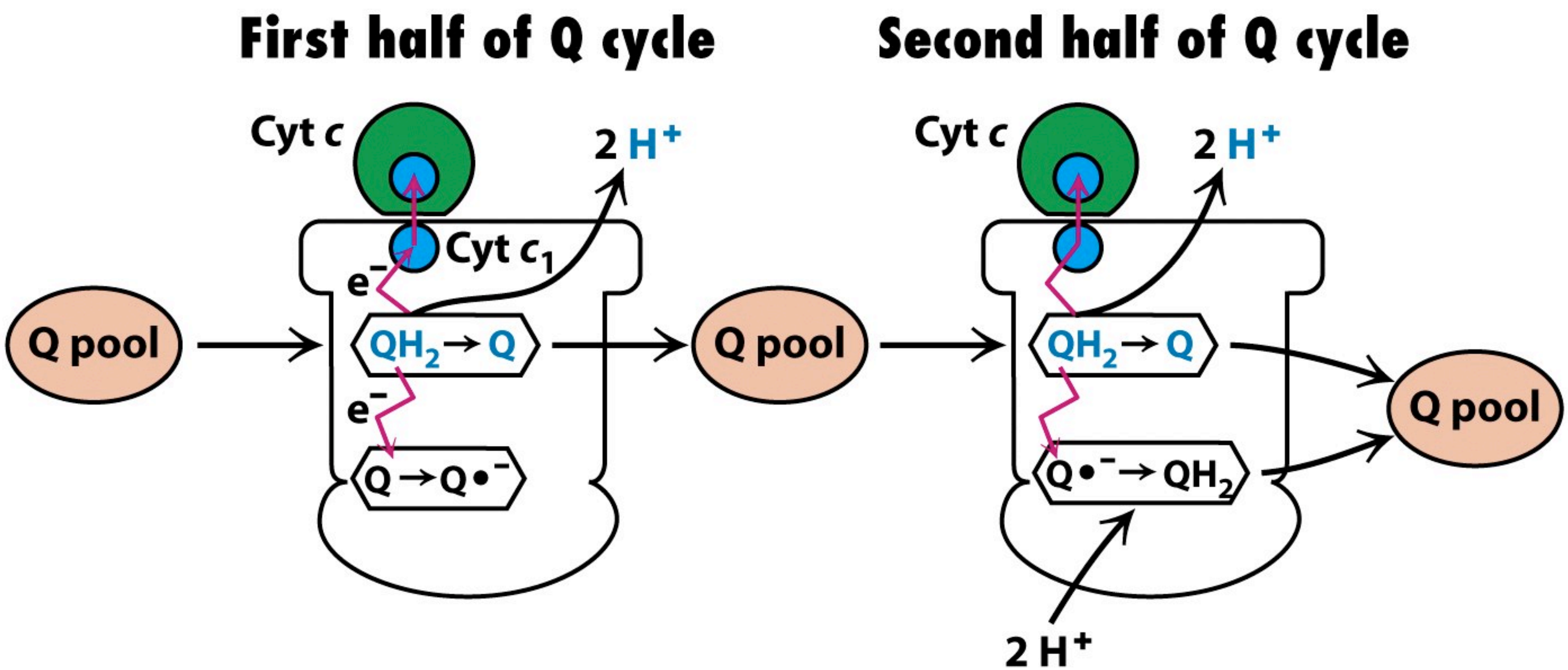


Figure 18-12
Biochemistry, Sixth Edition

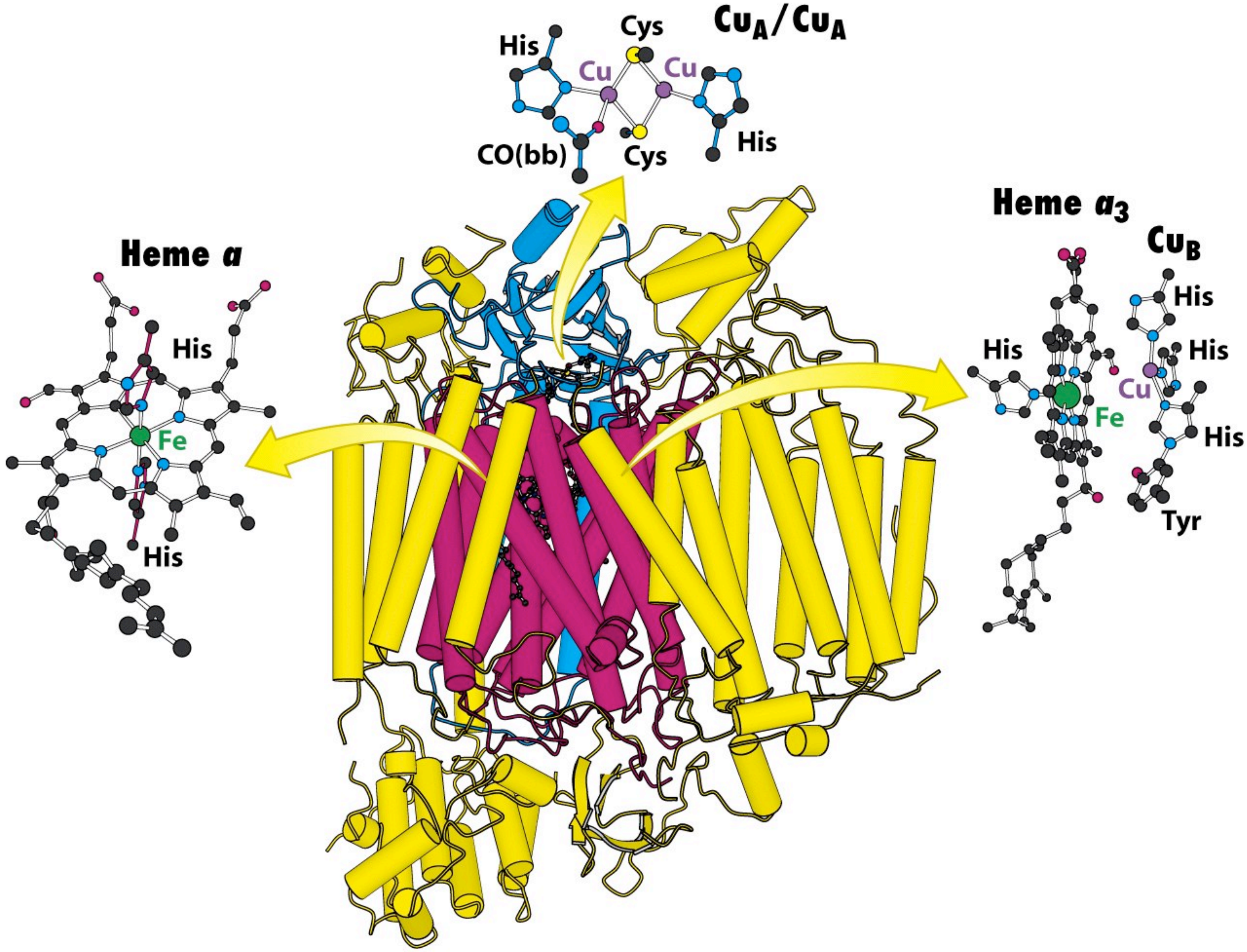


Figure 18-13
Biochemistry, Sixth Edition

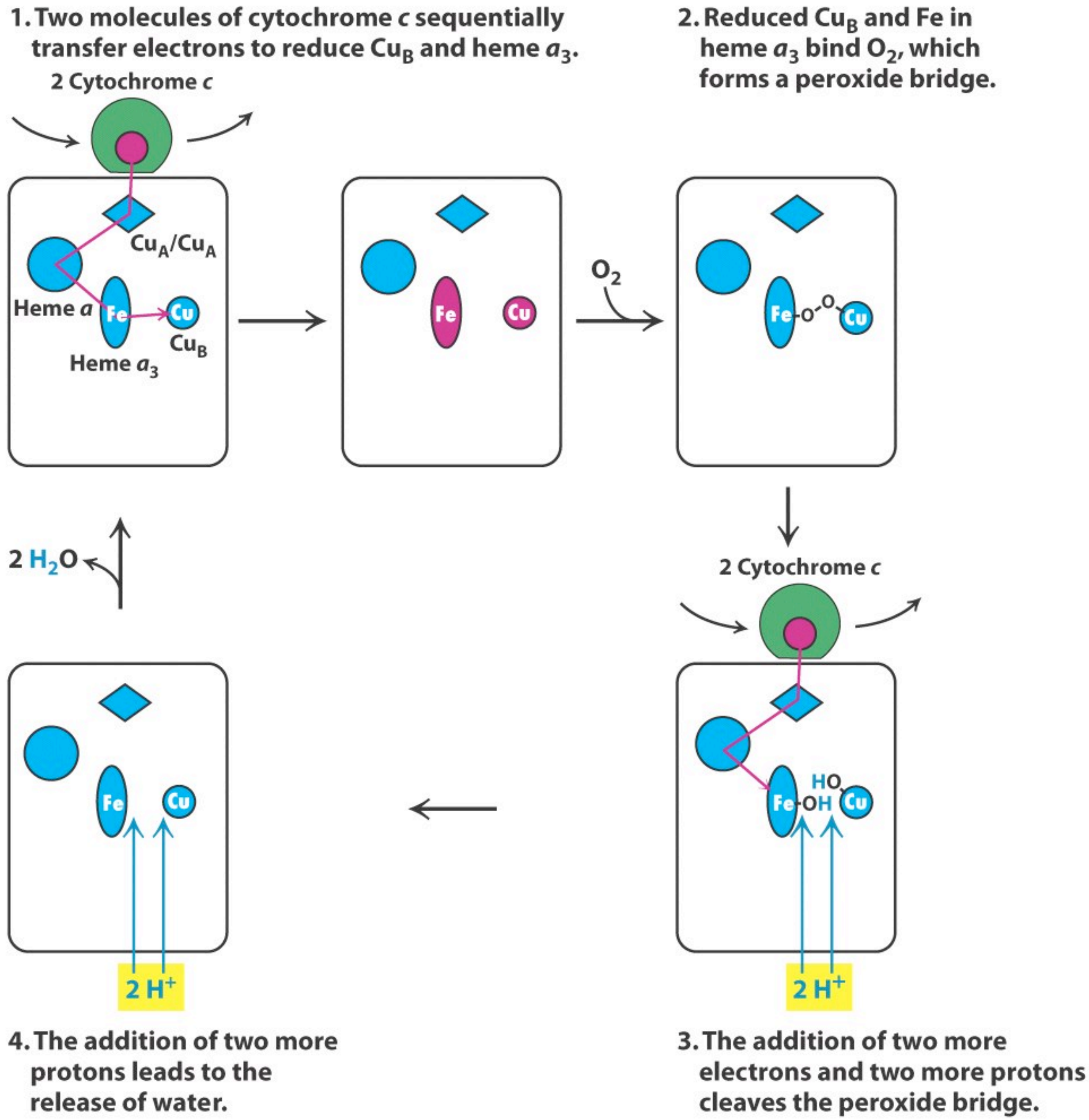


Figure 18-14
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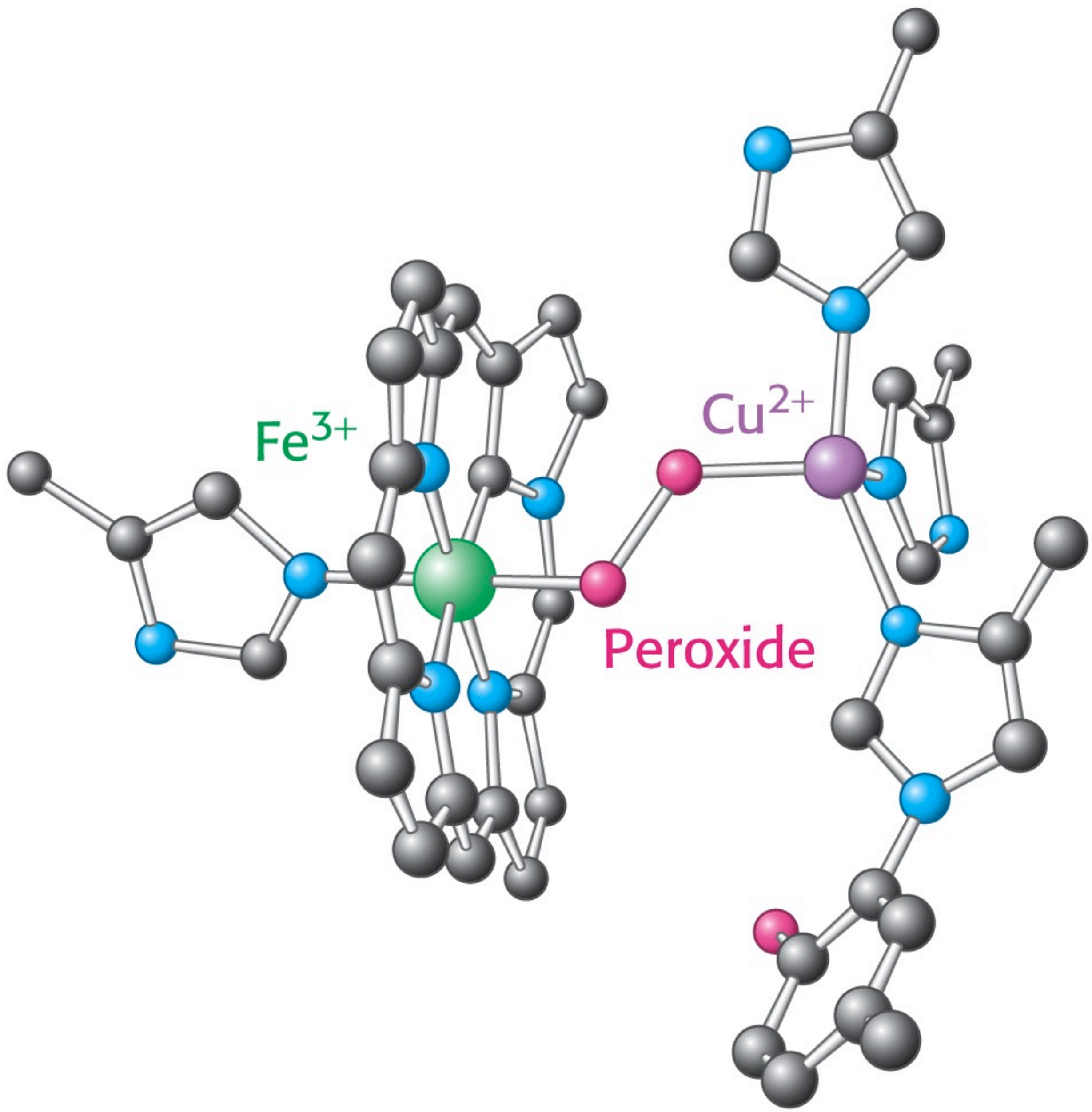

Fe^{3+}
Cu^{2+}
Peroxide

Overall

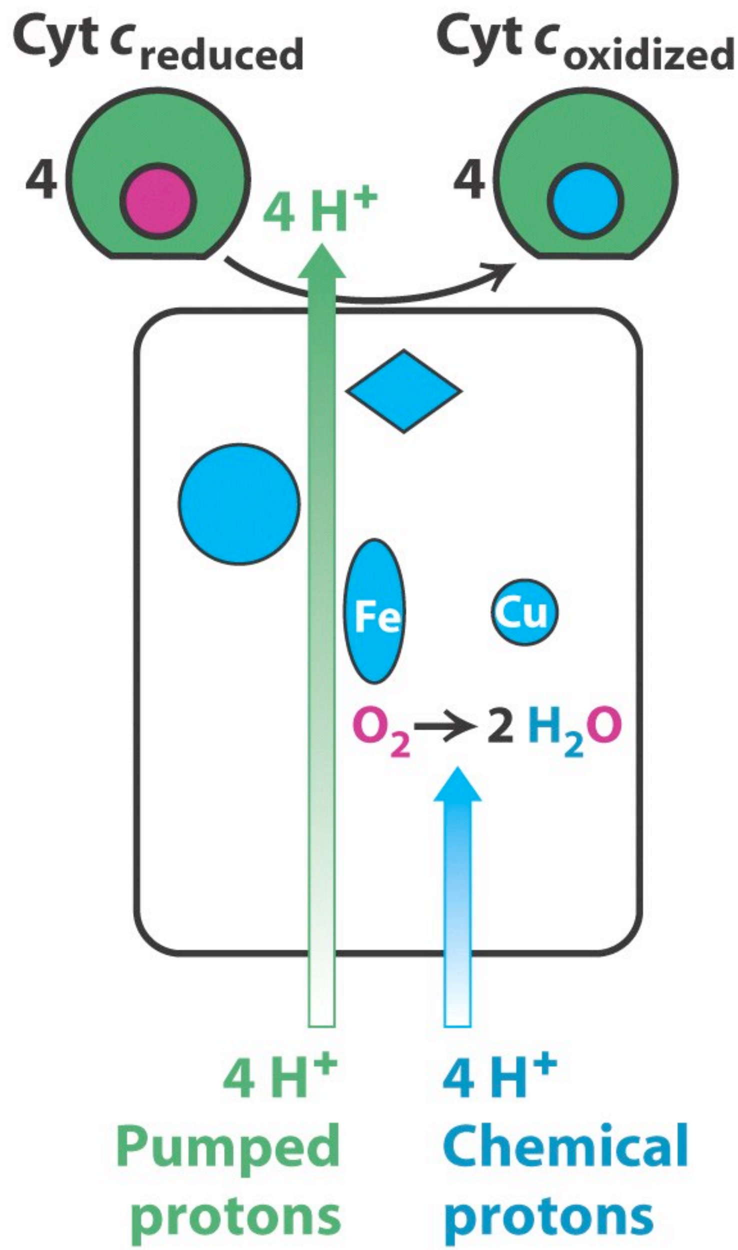

Figure 18-16
Biochemistry, Sixth Edition

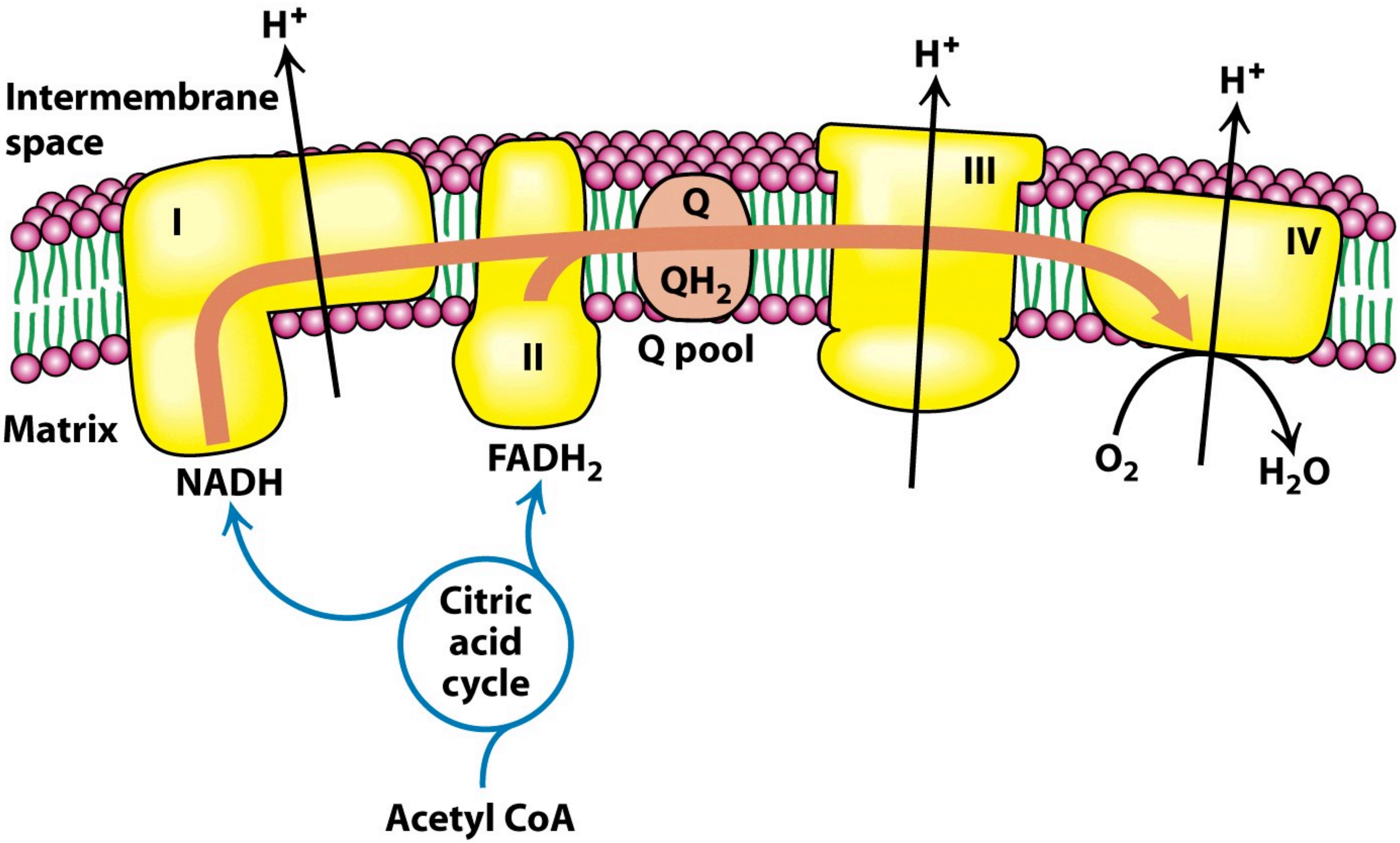

Figure 18-17
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The Danger of using Oxygen as an oxidizing agent !

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

Atherogenesis
Emphysema; bronchitis
Parkinson disease
Duchenne muscular dystrophy
Cervical cancer
Alcoholic liver disease
Diabetes
Acute renal failure
Down syndrome
Retrolental fibroplasia (conversion of the retina into a fibrous mass in premature infants)
Cerebrovascular disorders
Ischemia; reperfusion injury

Source: After D. B. Marks, A. D. Marks, and C. M. Smith, *Basic Medical Biochemistry: A Clinical Approach* (Williams & Wilkins, 1996), p. 331.

Table 18-3
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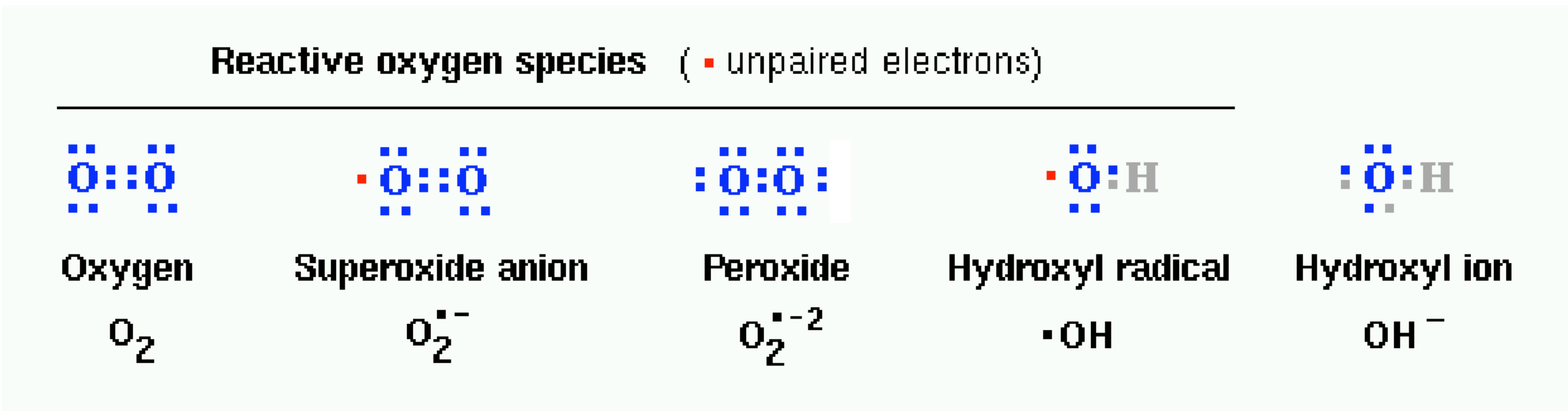
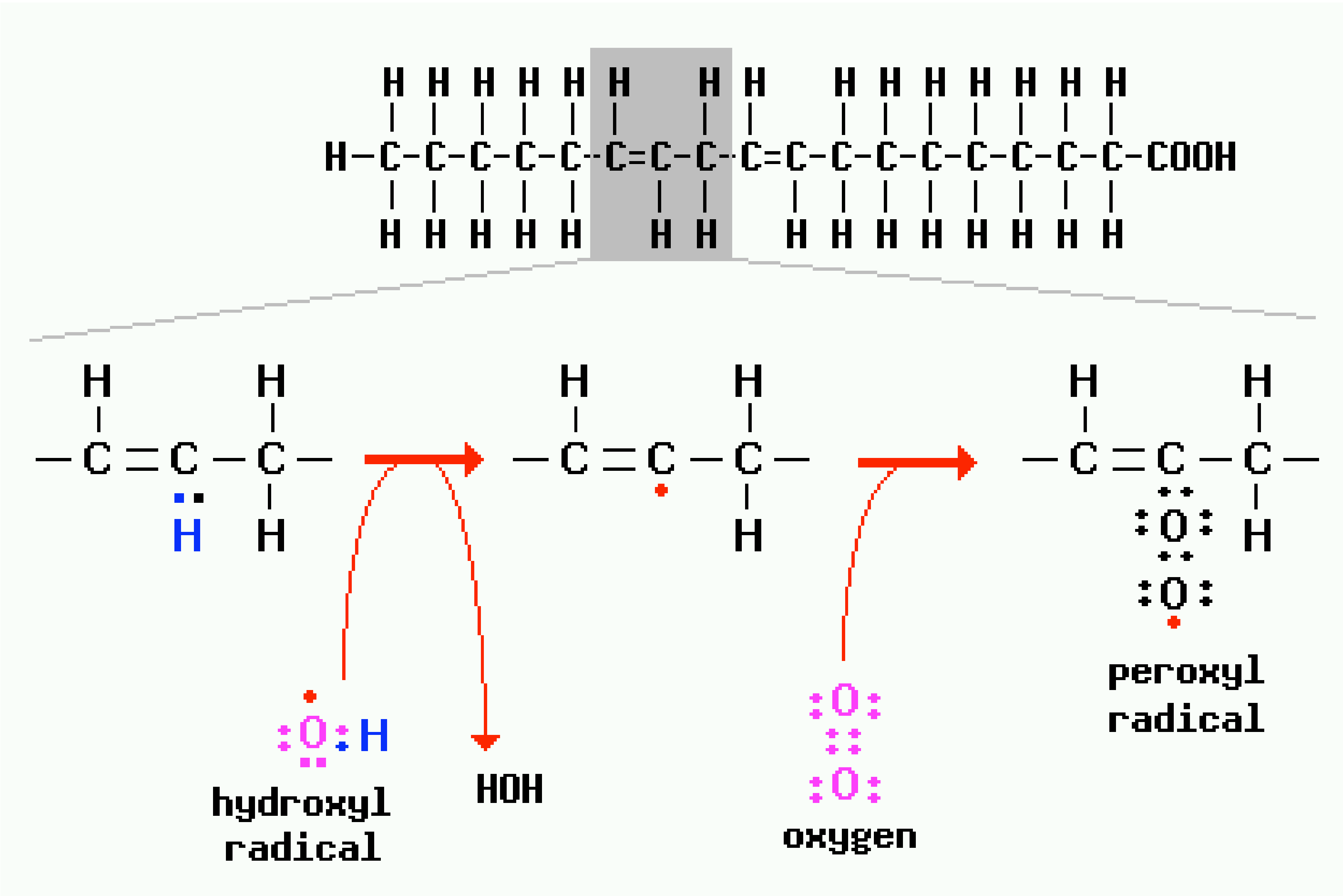
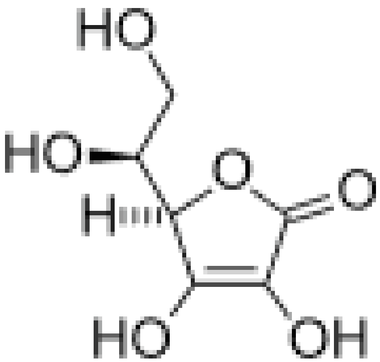
ROS


Reactive oxygen species (▪ unpaired electrons)
Oxygen
O_2
Superoxide anion
$O_2^{\bullet -}$
Peroxide
$O_2^{\bullet -2}$
Hydroxyl radical
$\bullet OH$
Hydroxyl ion
OH^-

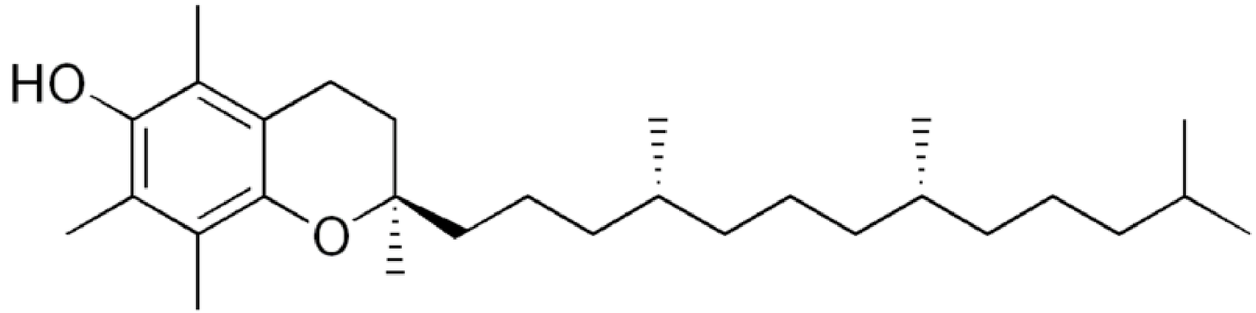
Oxygen Radical Damage



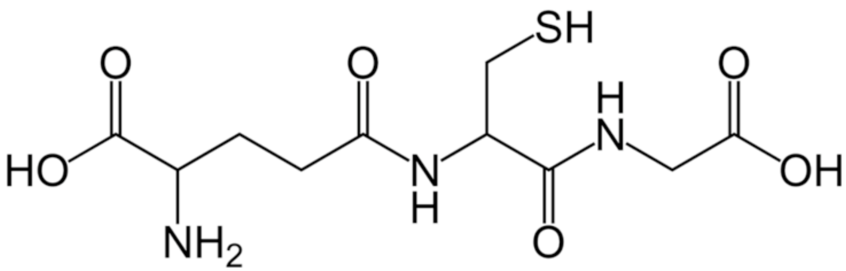
Oxygen Radical Scavengers: small molecules



Vitamin C

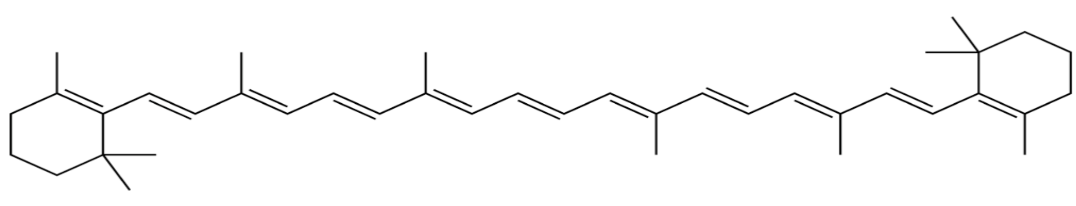


Vitamin e



glutathione

Beta carotene



Oxygen Radical Scavengers:Enzymes

- Superoxide dismutase

$$O_2^{\bullet -} + O_2^{\bullet -} \xrightarrow{\text{SOD}} O_2 + H_2O_2$$

(+2 H^+)

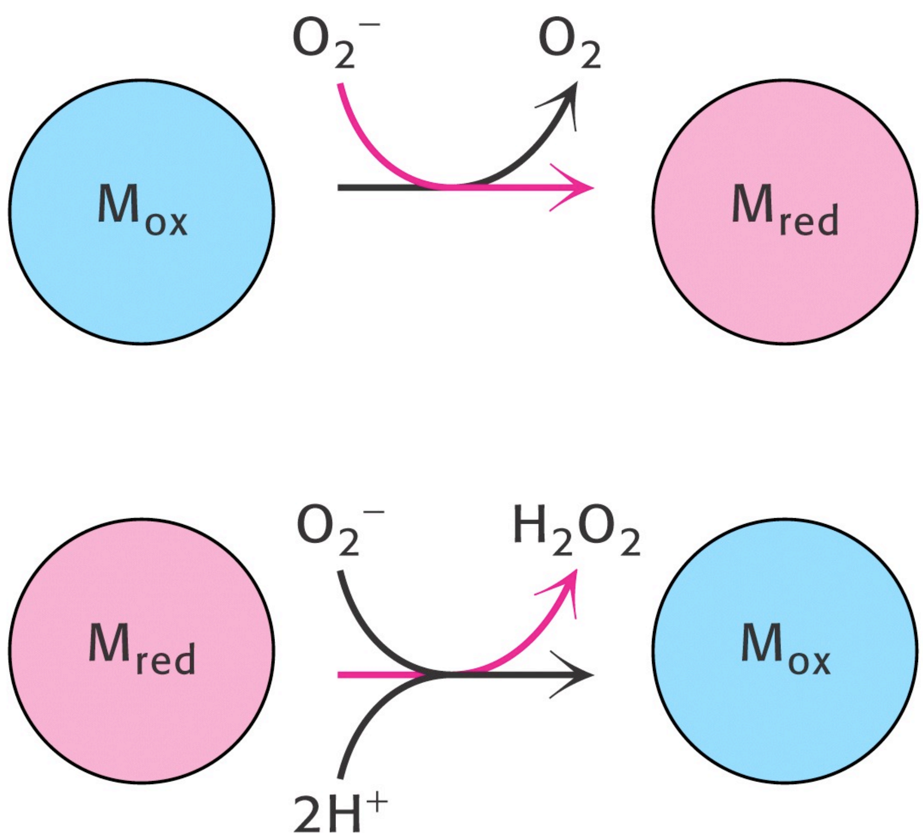
- Catalase: finishes the job

$$2H_2O_2 \rightarrow 2H_2O + O_2$$

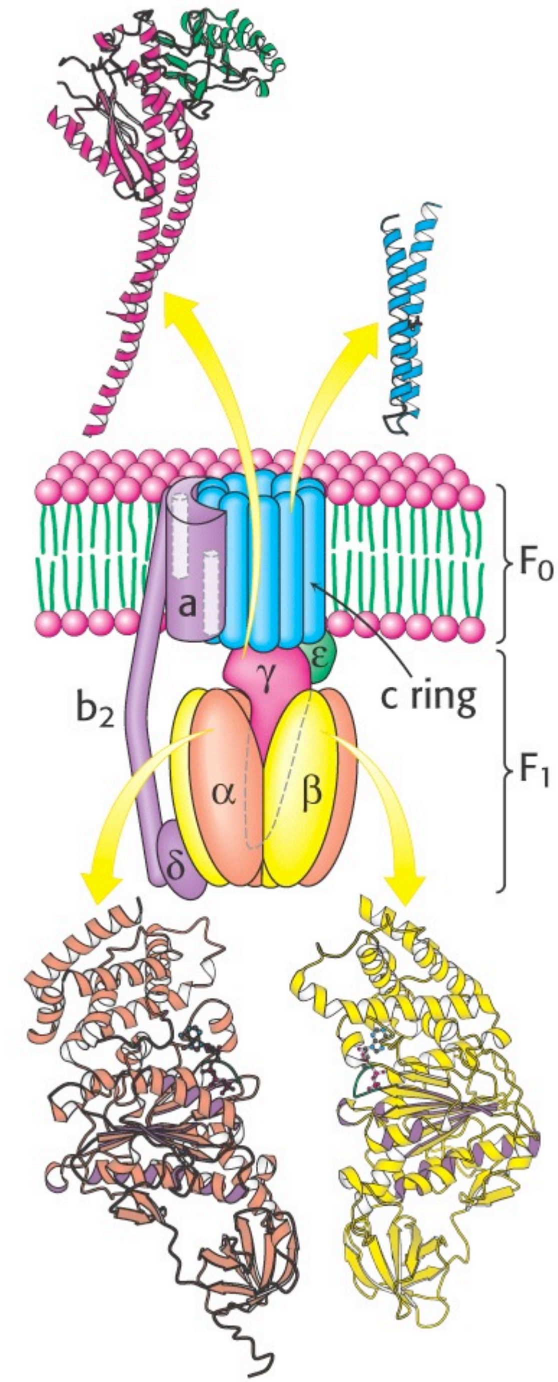
- Glutathione peroxidase

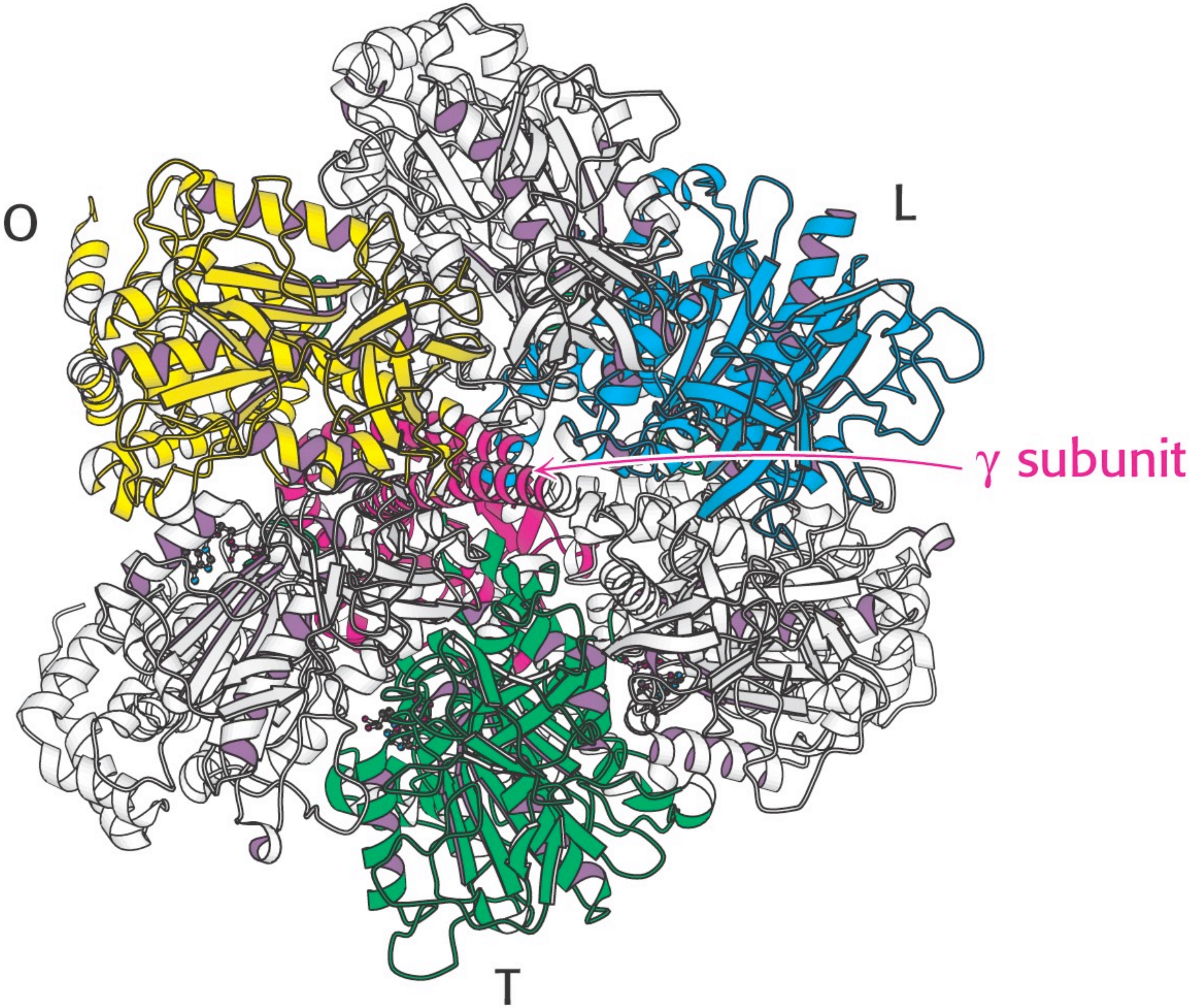
The Danger of using Oxygen as an oxidizing agent !

SOD takes off the Edge



The Final Payoff: the ATP ase




O
L
γ subunit
T

Boyer's Nobel Prize-winning model

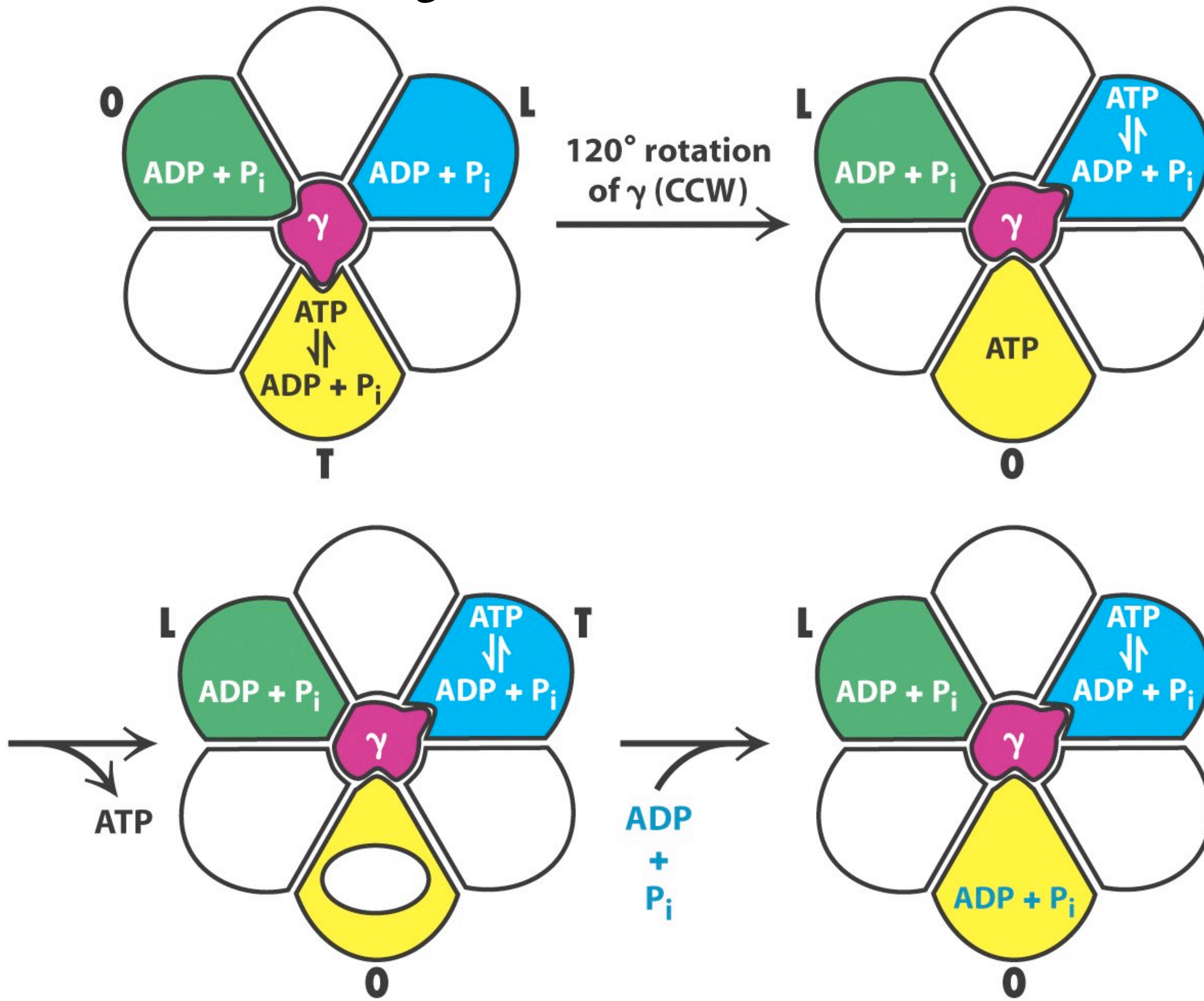


Figure 18-29
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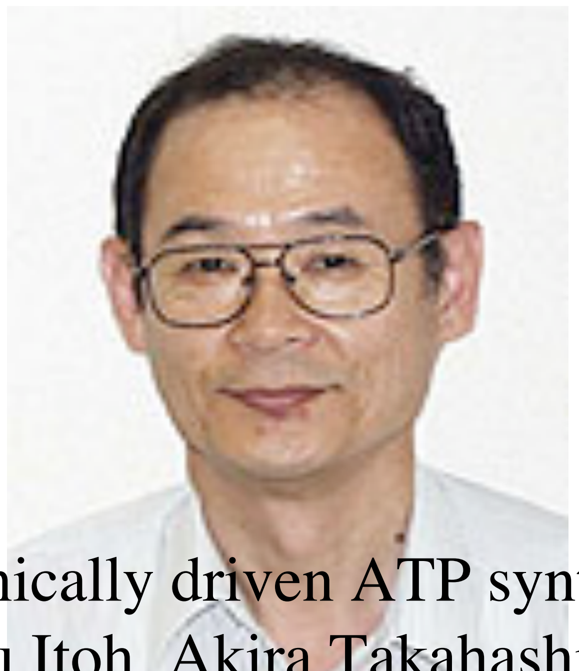
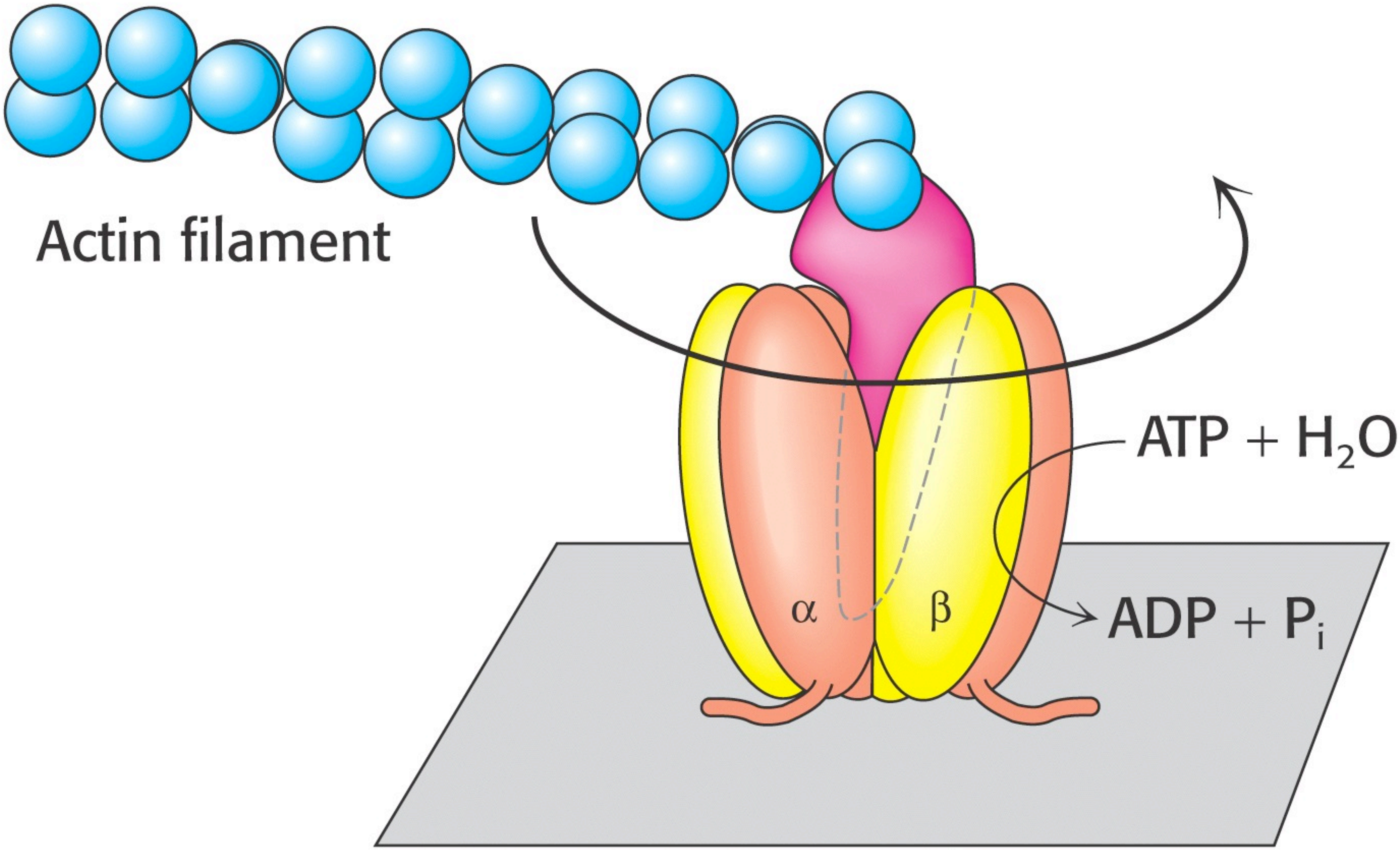
Probing Nature's Nanoscale Machines with Microscale Probes



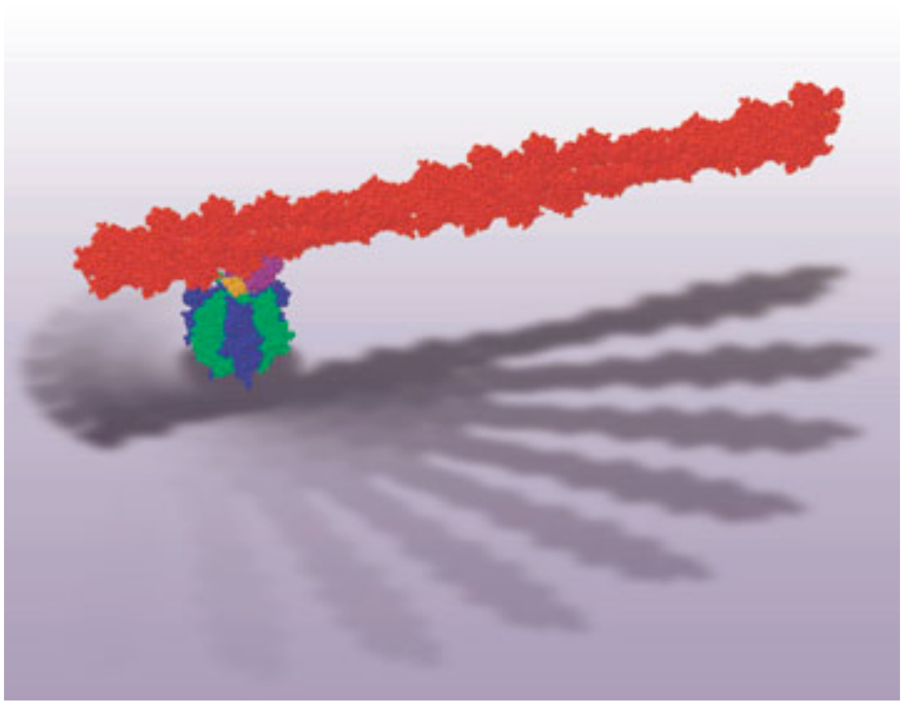
Kazuhiko Kinosita
Waseda University
Tokyo, Japan

"Mechanically driven ATP synthesis by F1-ATPase"
Hiroyasu Itoh, Akira Takahashi, Kengo Adachi, Hiroyuki Noji, Ryohei Yasuda, Masasuke Yoshida, and Kazuhiko Kinosita, Jr.
Nature, 427 (2004) 465-468.



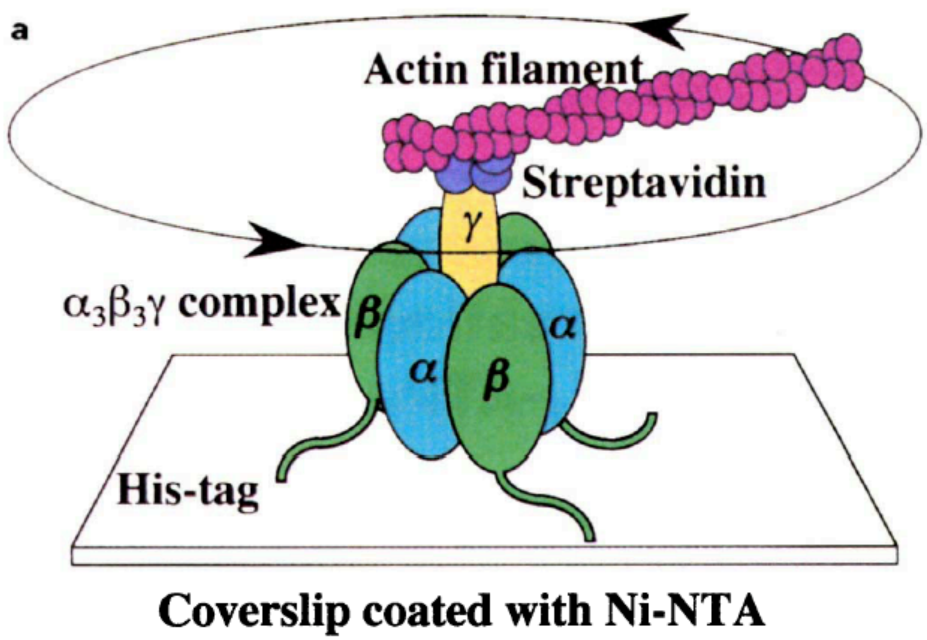
Actin filament
ATP + H_2O
ADP + P_i
α
β

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?

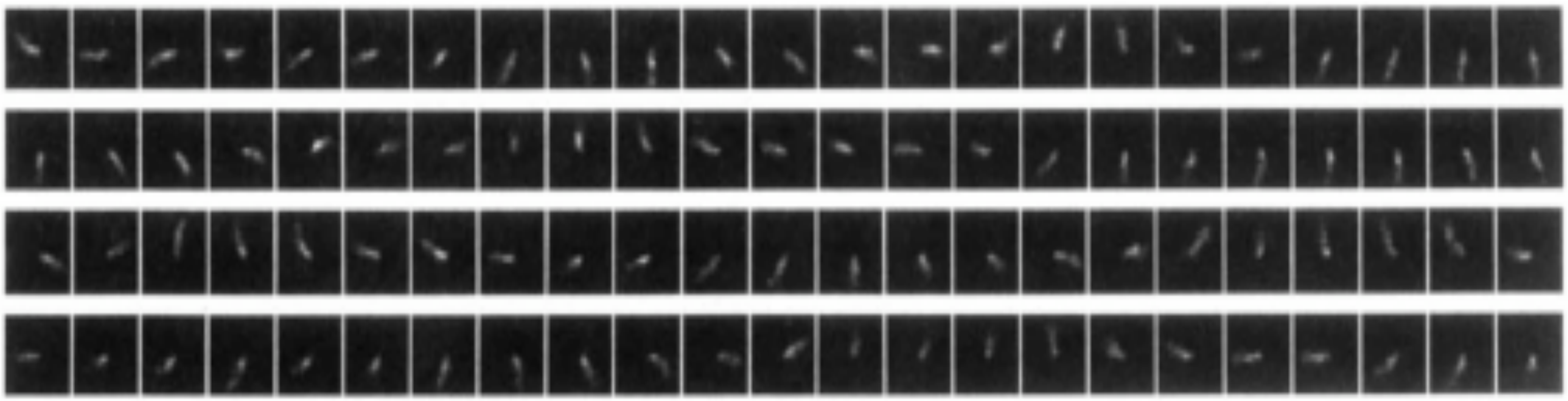


- The F1 complex from a thermophilic bacterium was expressed in E coli with 10 histidines linked to the N terminus of each beta subunit
- Glass plate was coated with horseradish peroxidase conjugated with Ni^{2+} nitrilotriacetic acid

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

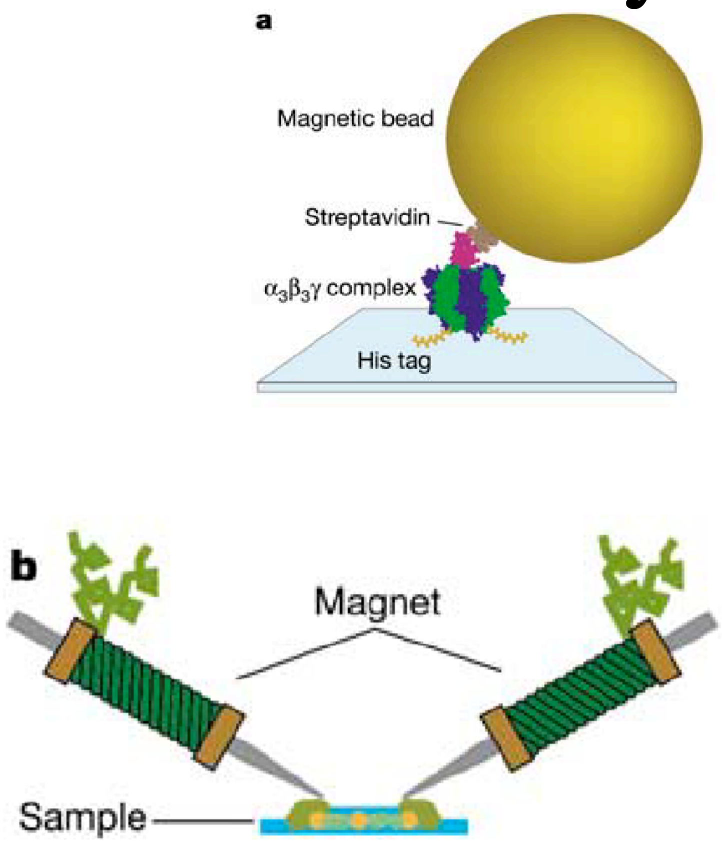
- Central rotor, the gamma subunit, is 1 nm in radius and the barrel in which it rotates, made up of the beta and alpha subunits, is 5nm in radius
- In the presence of 2 mM ATP, the F1 ATPase rotated the actin filament CCW 100+ revolutions and produced a constant torque of about 40 pN nm

Back to Kazuhiko Kinosita

"Coupling of rotation and catalysis in F1-ATPase revealed by single-molecule imaging and manipulation"
Kengo Adachi, Kazuhiro Oiwa, Takayuki Nishizaka, Shou Furuike, Hiroyuki Noji, Hiroyasu Itoh, Masasuke Yoshida, and Kazuhiko Kinosita Jr.
Cell 130 (2007) 309-321.

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

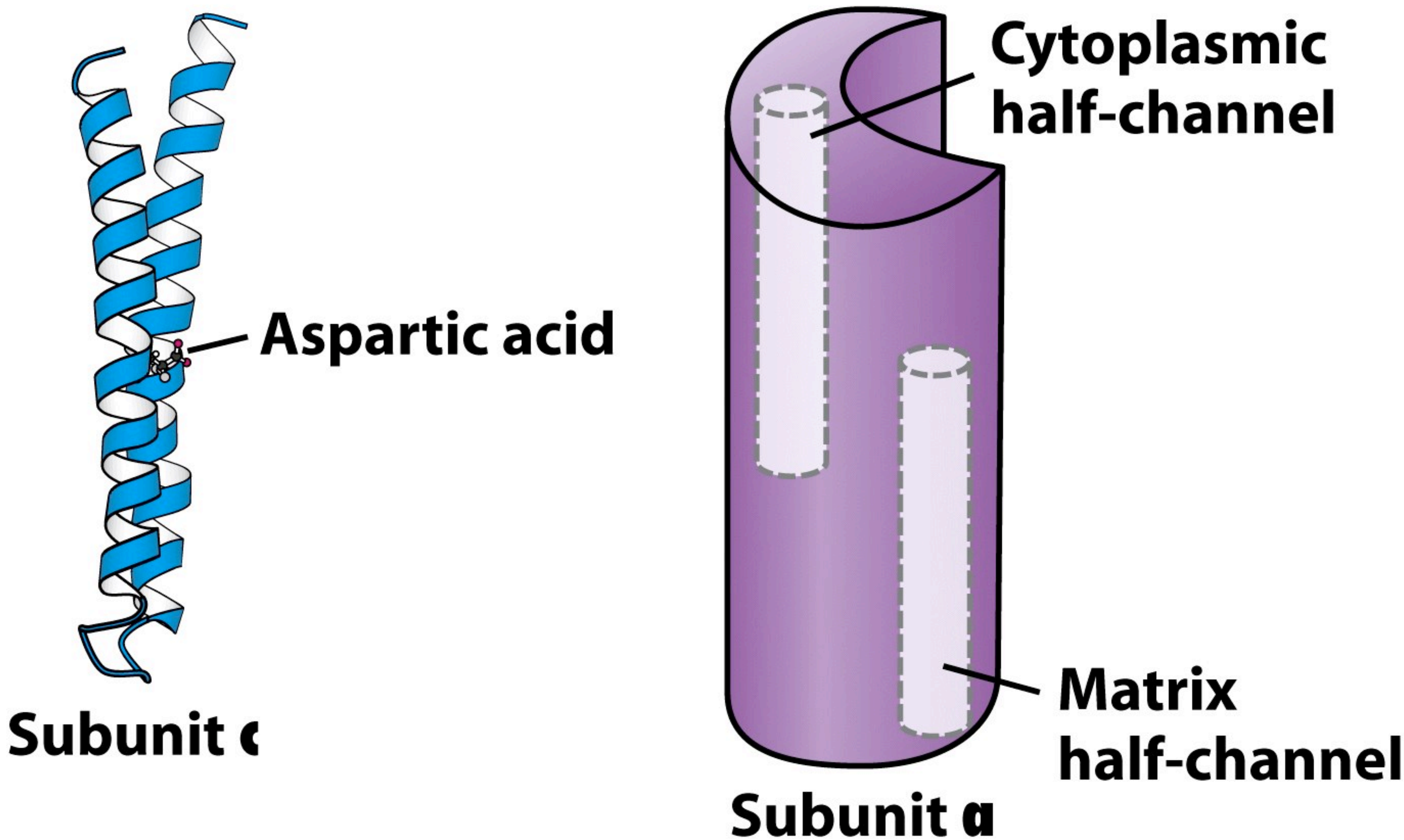


Figure 18-31
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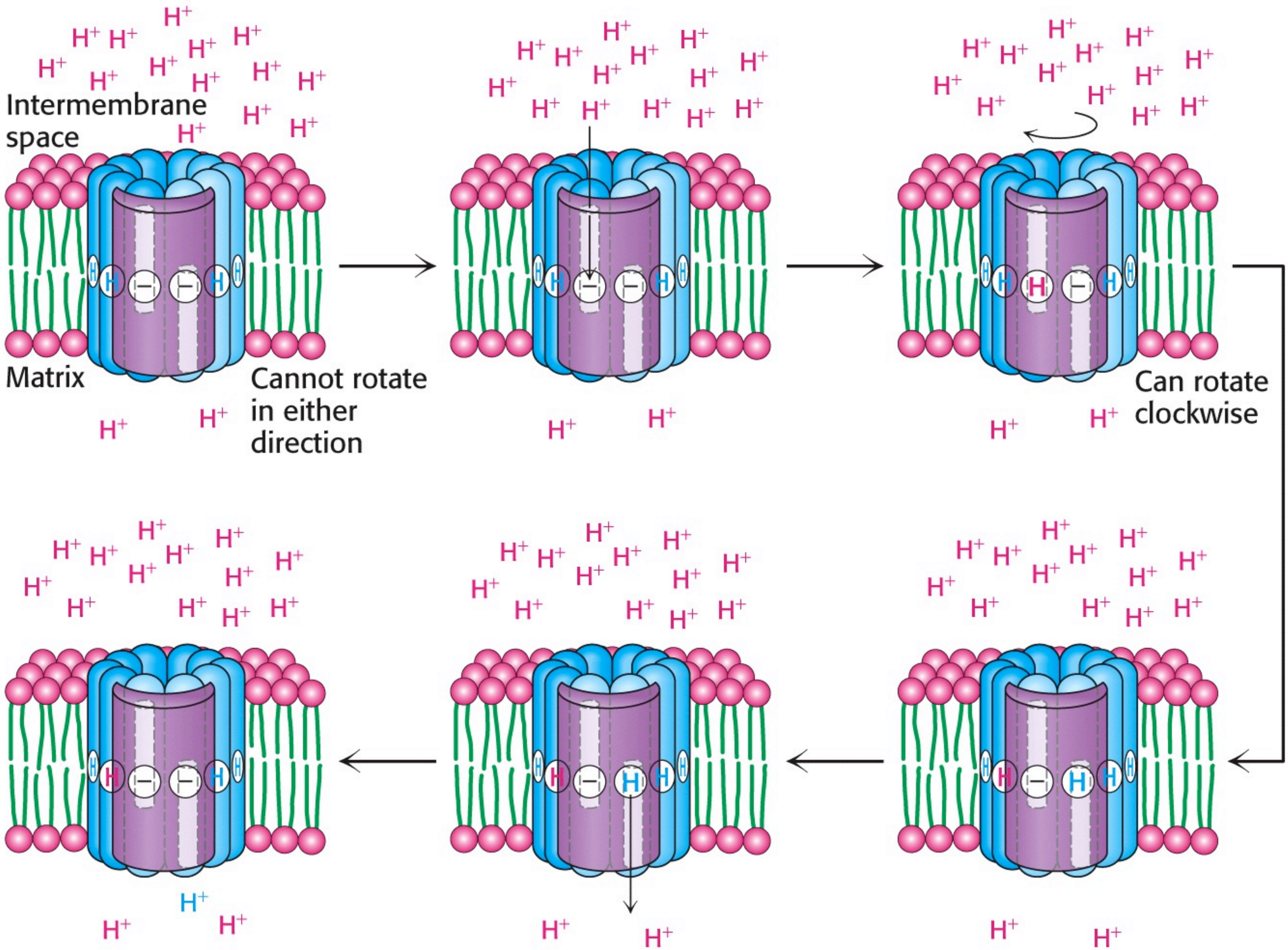
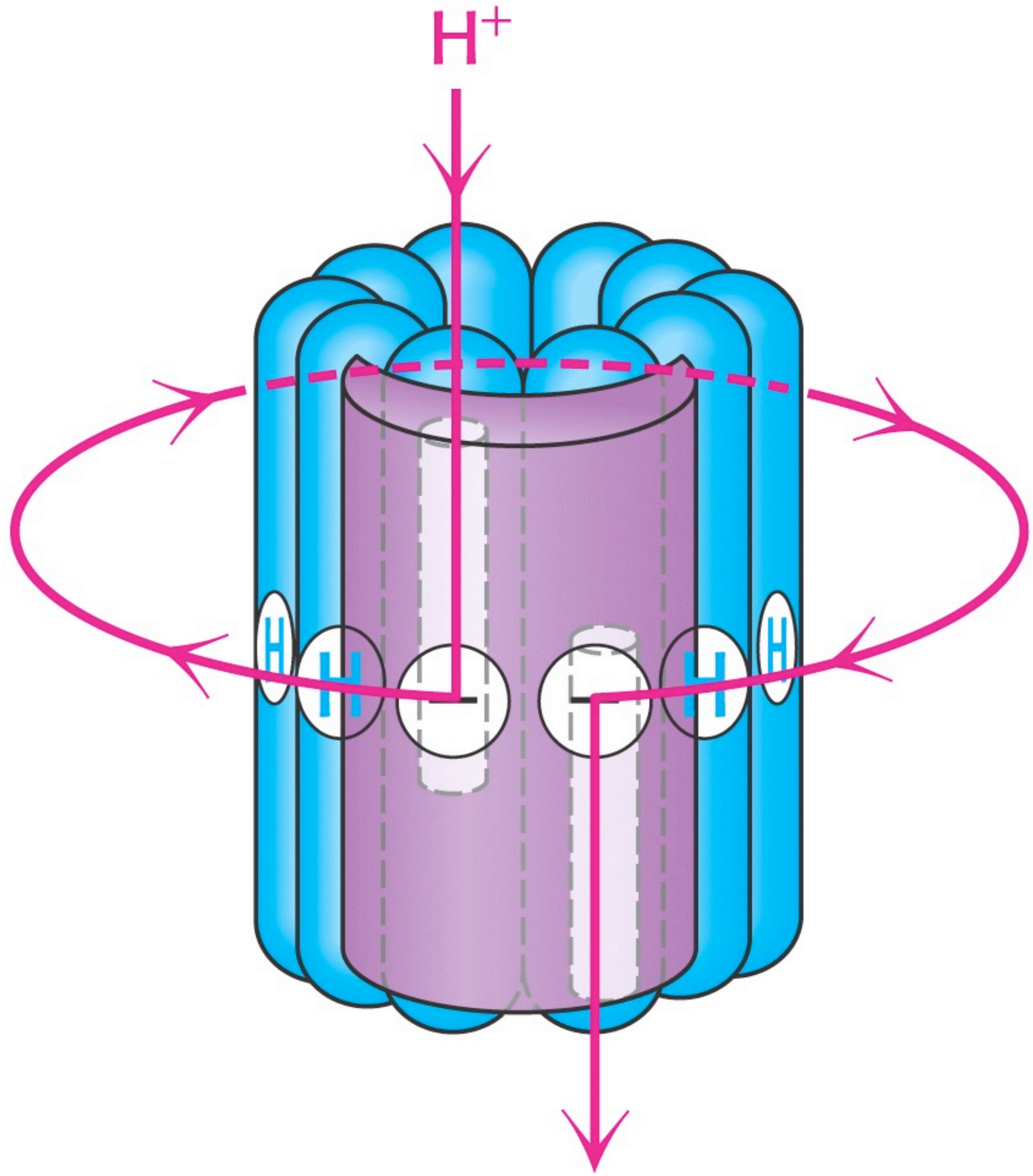
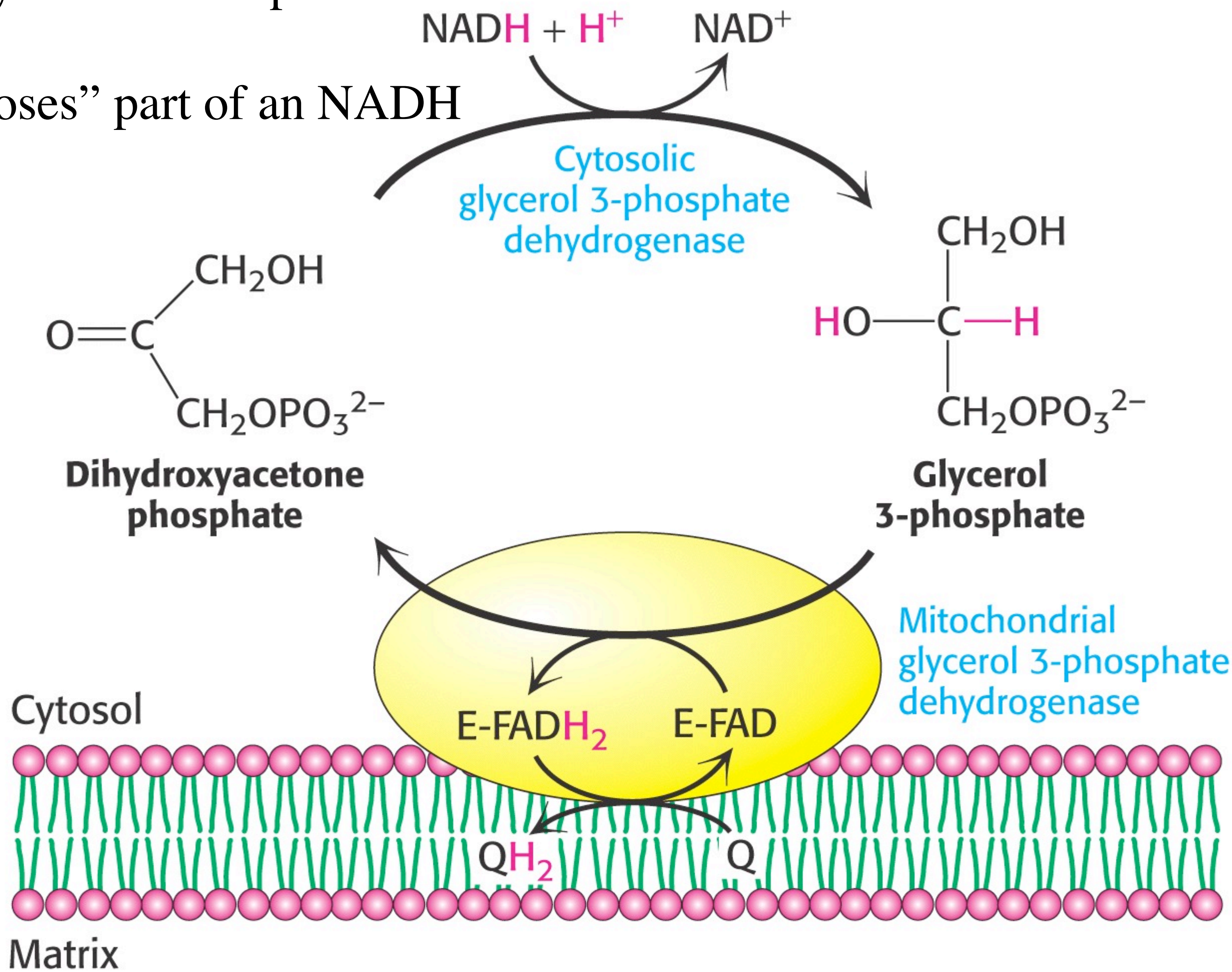

Intermembrane space
Matrix
Cannot rotate in either direction
Can rotate clockwise
H+


H+

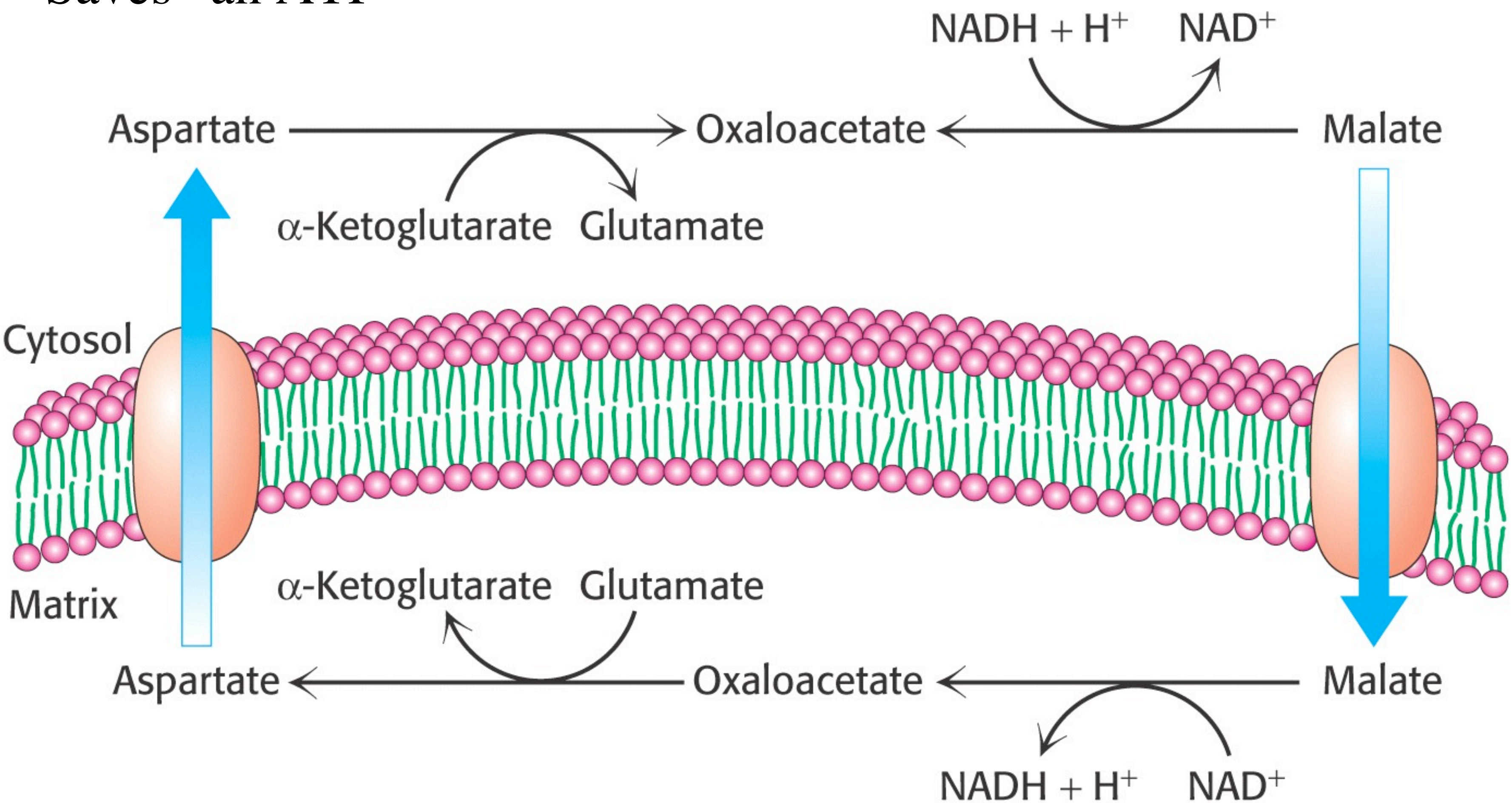
Glycerol-3-Phosphate shuttle

“Loses” part of an NADH



Malate/Aspartate shuttle-in heart and liver

“Saves” an ATP



Getting it out! The ATP translocase

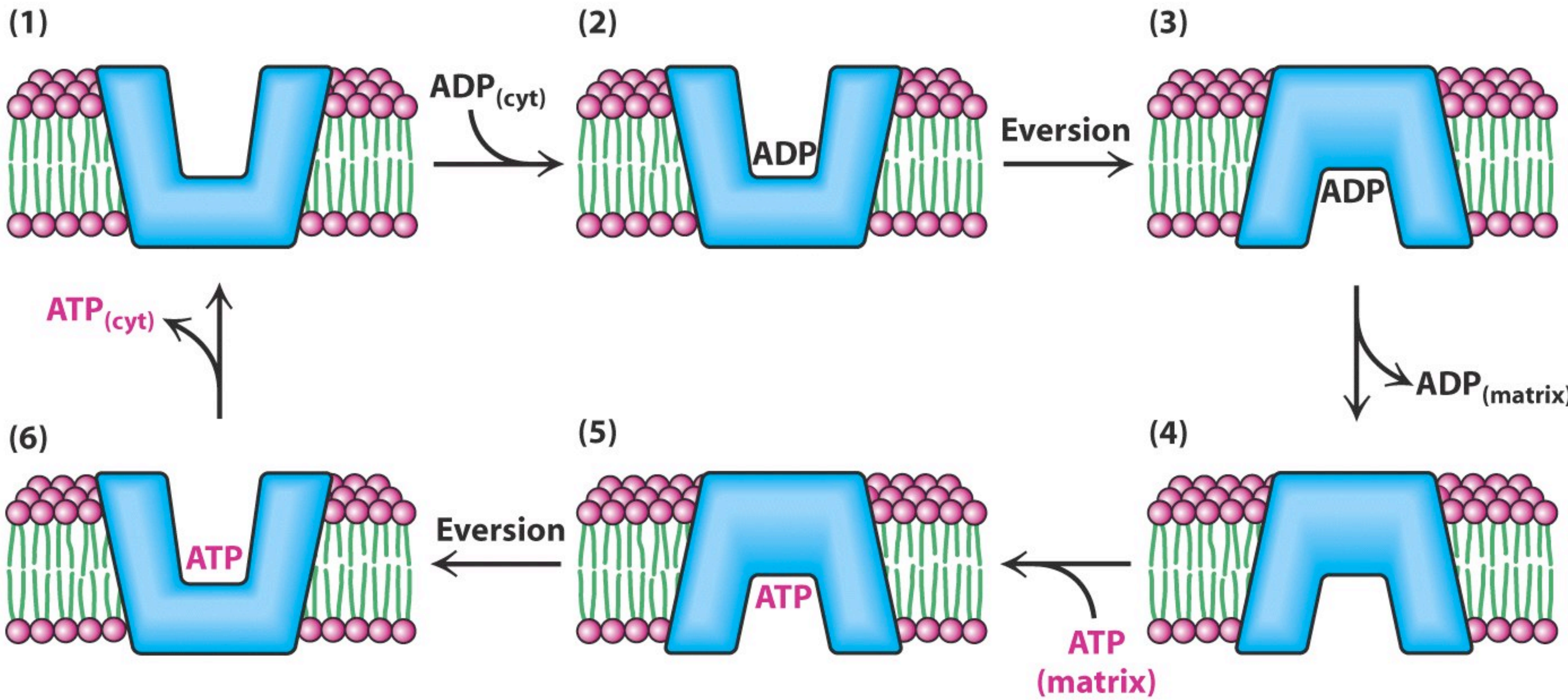


Figure 18-37
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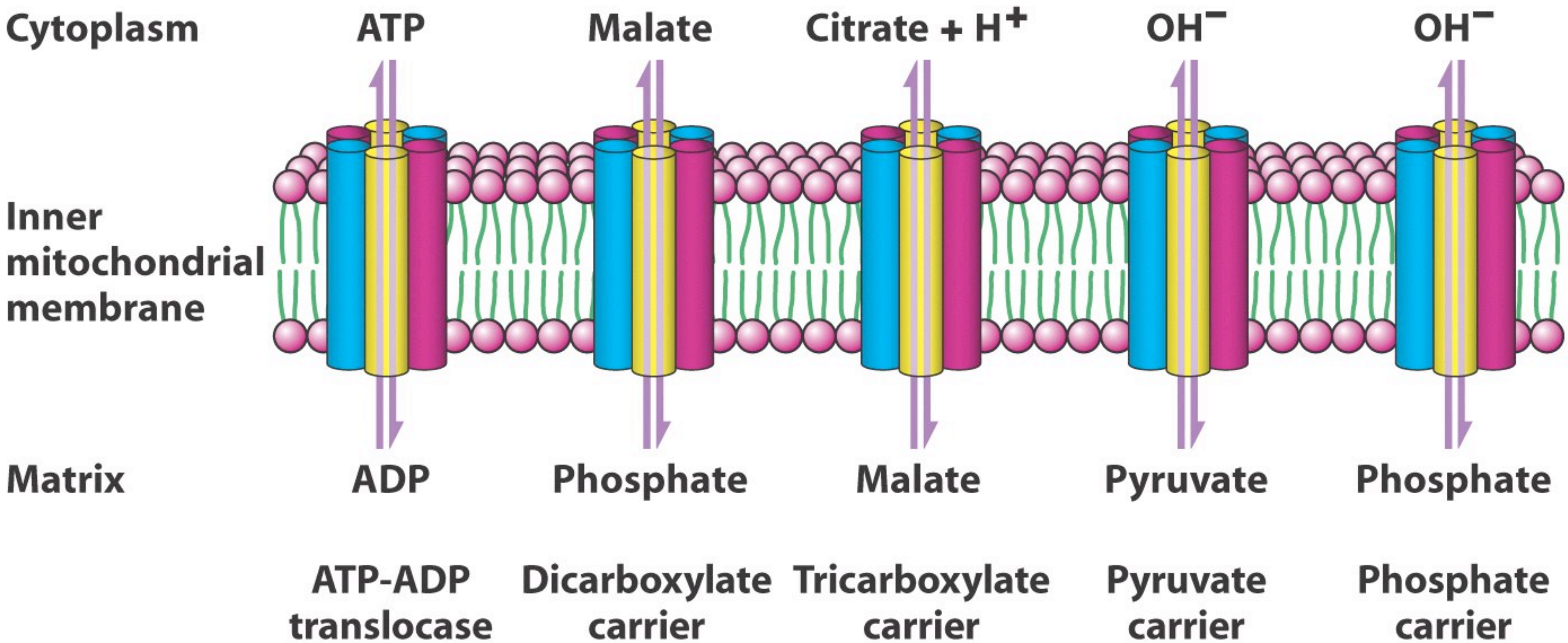

Figure 18-39
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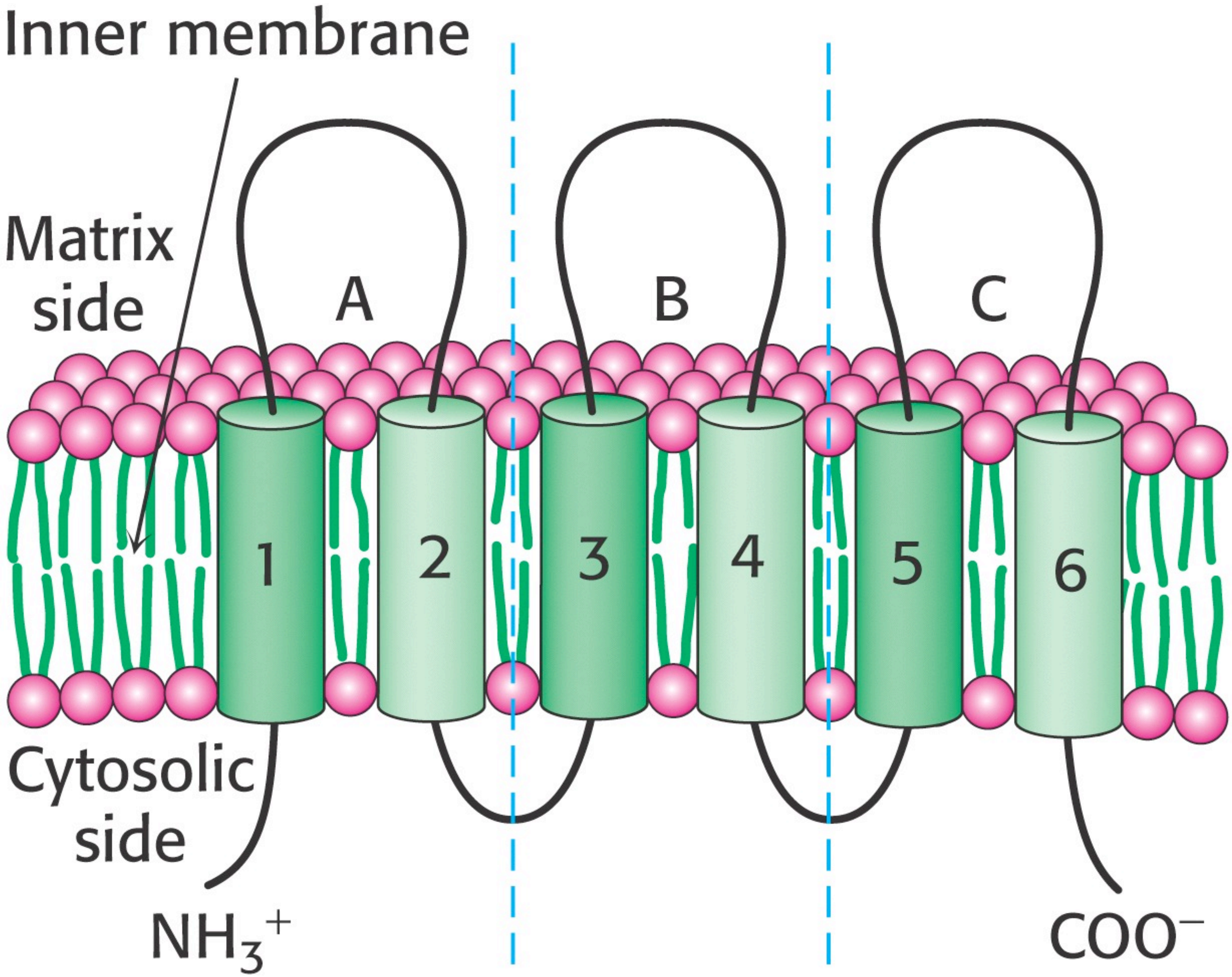
Common Structure of Mito Transporters



Problem

For, summing up synthesis of **~P bonds** via oxidative phosphorylation, assume:

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

1.5 ~P bonds synthesized per **NADH** produced in the cytosol in **Glycolysis** (electrons transferred via FAD to coenzyme Q).

1.5 ~P bonds synthesized during oxidation of **$FADH_2$** produced in **Krebs Cycle** (Succinate Dehydrogenase – electrons transferred to coenzyme Q).

All Quantities Per Glucose

Pathway	NADH produced	$FADH_2$ produced (QH_2)	~P bonds ATP or GTP direct	~P bonds 1.5 or 2.5 per NADH in oxphos	~P bonds 1.5 per $FADH_2$ in oxphos	Total ~P bonds
Glycolysis Pathway						
Pyruvate Dehydrogenase						
Krebs Cycle						
Sum of Pathways						

Table 18.4 ATP yield from the complete oxidation of glucose

Reaction sequence	ATP yield per glucose molecule
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 molecules of guanosine triphosphate are formed from 2 molecules of succinyl CoA	+2
6 molecules of NADH are formed in the oxidation of 2 molecules each of isocitrate, α-ketoglutarate, and malate	
2 molecules of $FADH_2$ are formed in the oxidation of 2 molecules of succinate	
Oxidative phosphorylation (inside mitochondria)	
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

Source: The ATP yield of oxidative phosphorylation is based on values given in P. C. Hinkle, M. A. Kumar, A. Resetar, and D. L. Harris. *Biochemistry* 30(1991):3576.
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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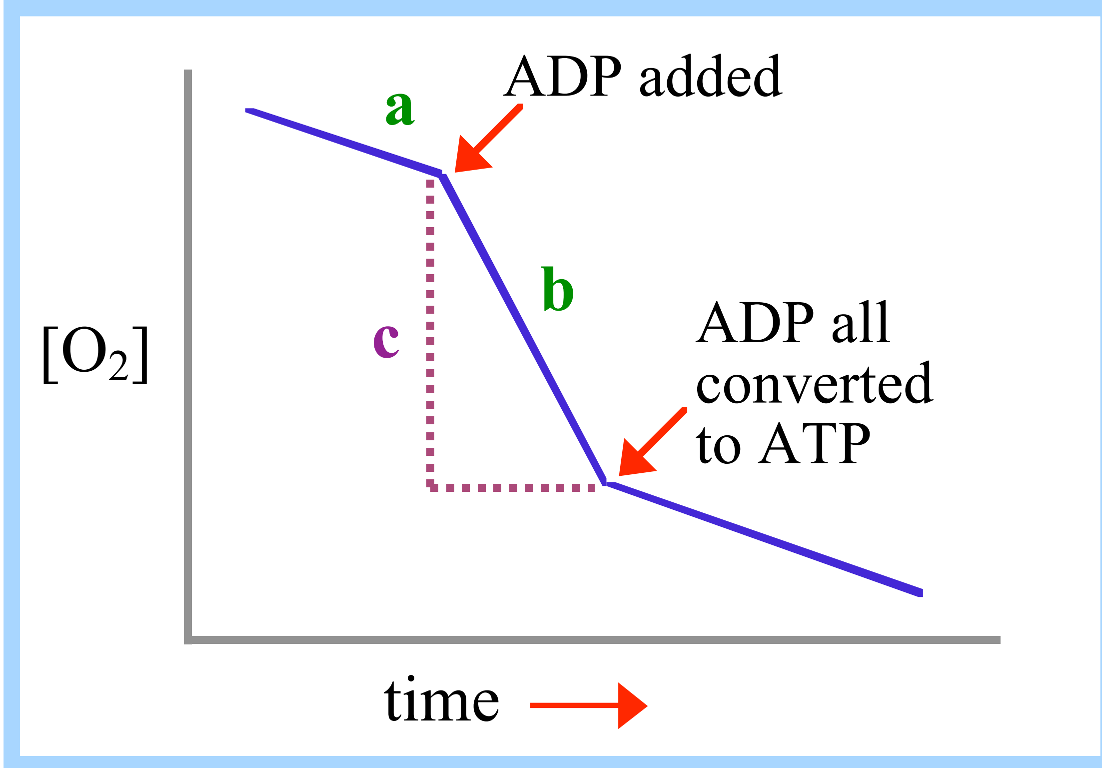
An **oxygen electrode** may be used to record [O_2] in a closed vessel.

Electron transfer, e.g., NADH → O_2, is monitored by the rate of O_2 disappearance.



Above is represented an O_2 electrode recording while mitochondria respire in the presence of P_i and an e^- donor (succinate or a substrate of a reaction to generate NADH).

The dependence of respiration rate on availability of 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.

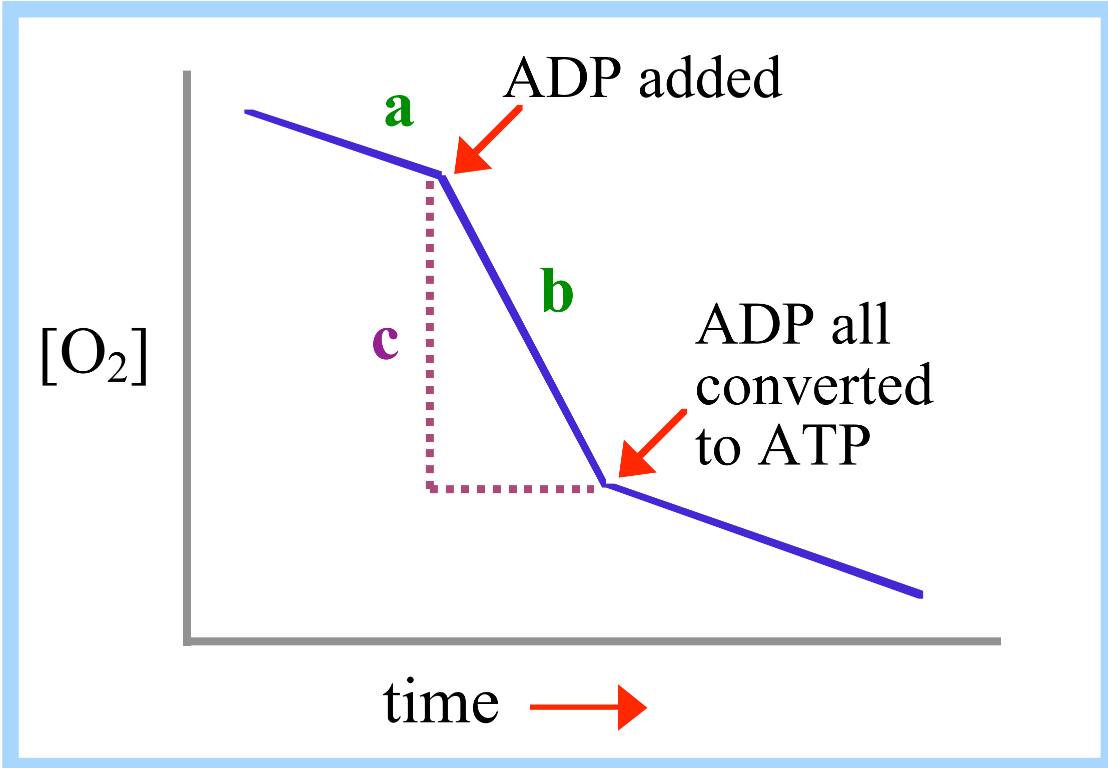
Reaction	ΔG
e^- transfer (NADH → O_2)	negative value*
H^+ ejection from matrix	positive; depends on H^+ gradient**
e^- transfer with H^+ ejection	algebraic sum of above

*$\Delta G^{o\prime} = -nF\Delta E^{o\prime} = -218$ kJ/mol for 2 e^- NADH → O_2.

**For ejection of 1 H^+ from the matrix:

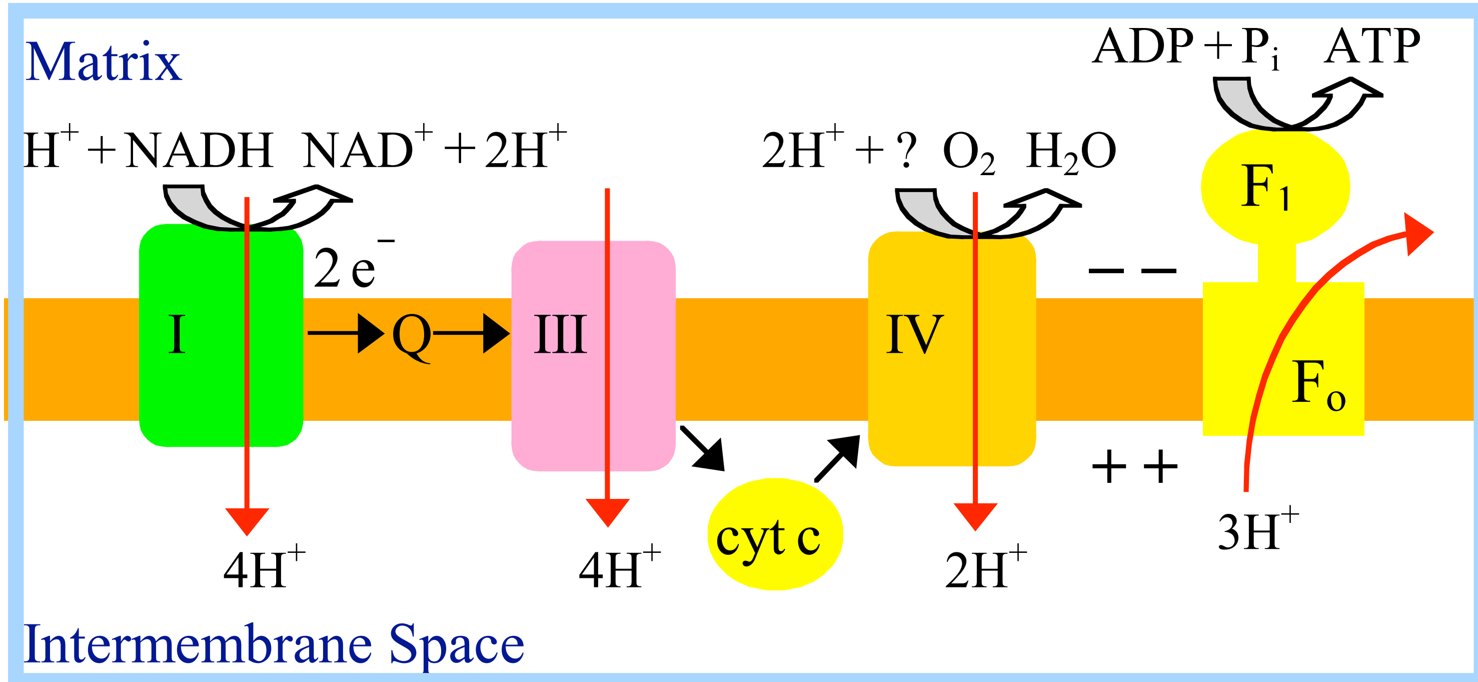
$$\Delta G = RT \ln ([H^+]_{cytosol}/[H^+]_{matrix}) + F\Delta\Psi$$

$$\Delta G = 2.3\ RT\ (pH_{matrix} - pH_{cytosol}) + F\Delta\Psi$$



Respiratory control ratio is the ratio of slopes after and before ADP addition (**b/a**).

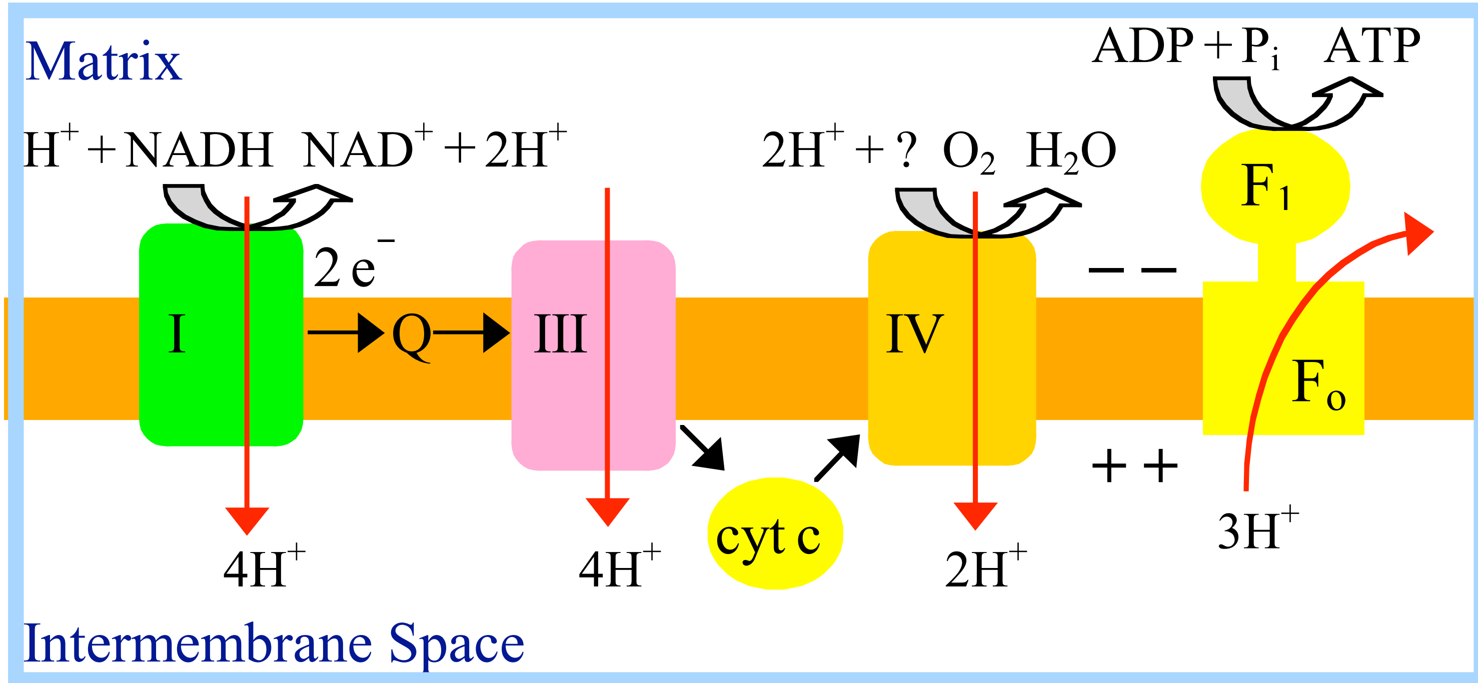
P/O ratio is the moles of ADP divided by the moles of O consumed (based on **c**) while phosphorylating the ADP.



With **no ADP**, H^+ cannot flow through F_o. ΔpH & $\Delta\Psi$ 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 ΔpH & ΔΨ.

ΔG 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

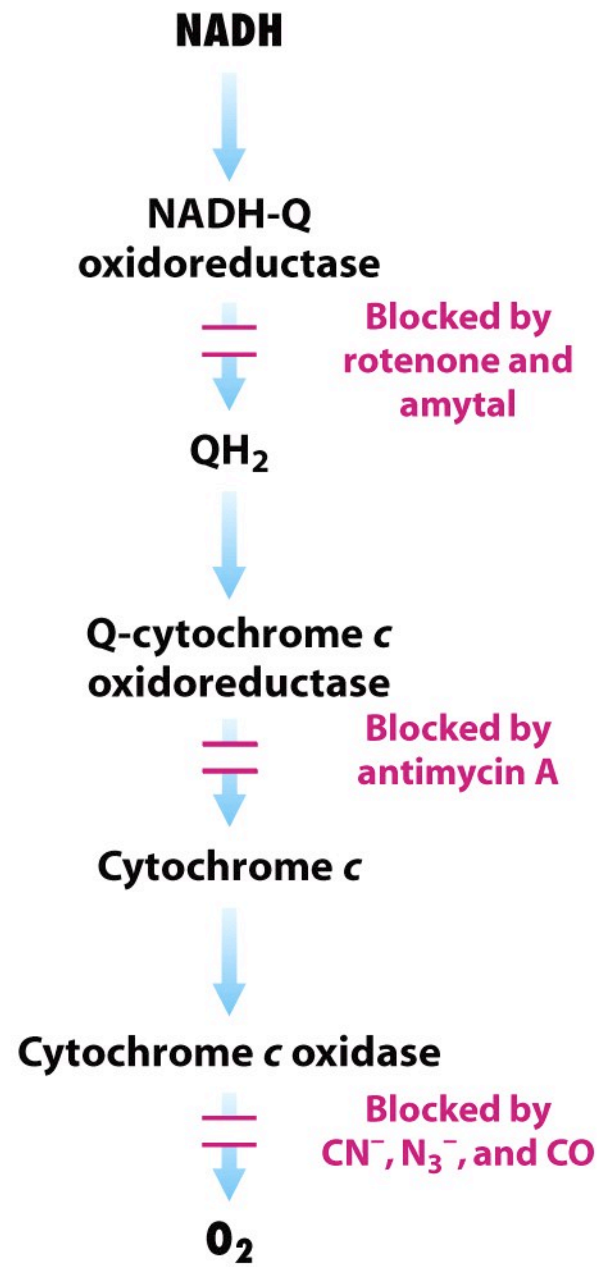


Figure 18-43
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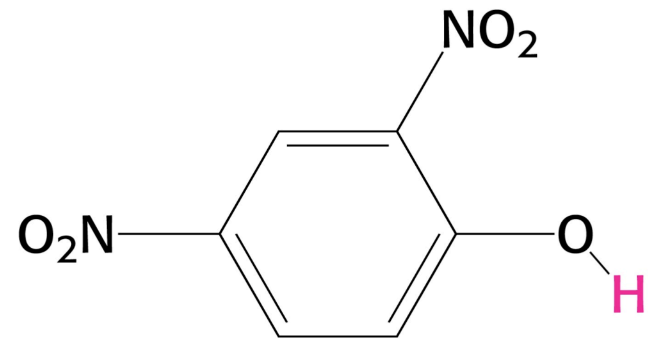
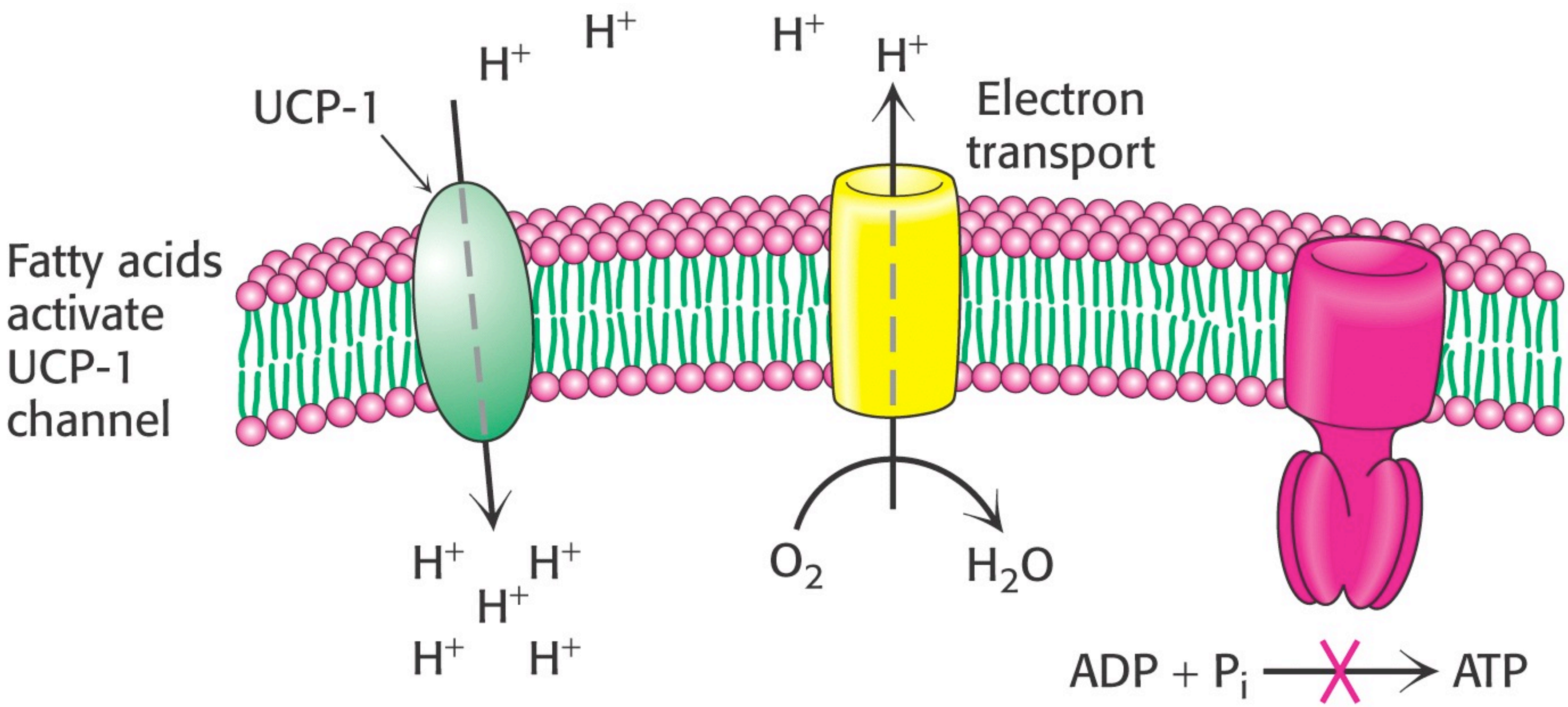
Uncouplers: Mitchell's revenge!!



2,4-Dinitrophenol (DNP)



Uncouplers: Mitchell's revenge!!

Regulatory Toxicology and Pharmacology
Volume 48, Issue 2, July 2007, Pages 115-117

Abstract | Full Text + Links | PDF (106 K)

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doi:10.1016/j.yrtph.2007.03.006 Cite or Link Using DOI
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Commentary

Dinitrophenol and obesity: An early twentieth-century regulatory dilemma☆

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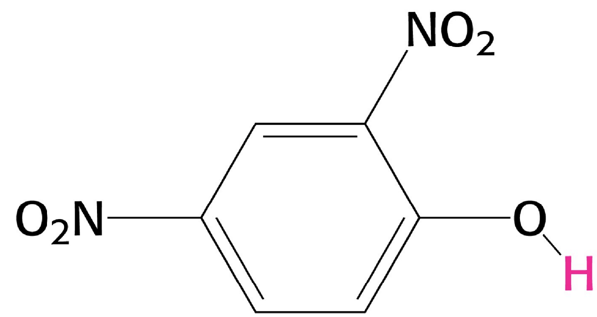
[a]Division of Metabolism and Endocrinology Products, Office of Drug Evaluation II, Center for Drug Evaluation and Research, U.S. Food and Drug Administration, 10903 New Hampshire Avenue, Building 22, Room 3360, Silver Spring, MD 20993, USA
Received 14 March 2007. Available online 31 March 2007.

Abstract

In the early 1930s, the industrial chemical dinitrophenol found wides
drug, due principally to the work of Maurice Tainter, a clinical pharma
University. Unfortunately the compound's therapeutic index was razo
thousands of people suffered irreversible harm that mainstream phys
dinitrophenol's risks outweighed its benefits and abandoned its use.
Food, Drug, and Cosmetic Act in 1938 before federal regulators had
medicine men from selling dinitrophenol to Americans lured by the p
safely melt one's fat away.



2,4-Dinitrophenol (DNP)

1: Obes Rev. 2001 Nov;2(4):255-65.

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

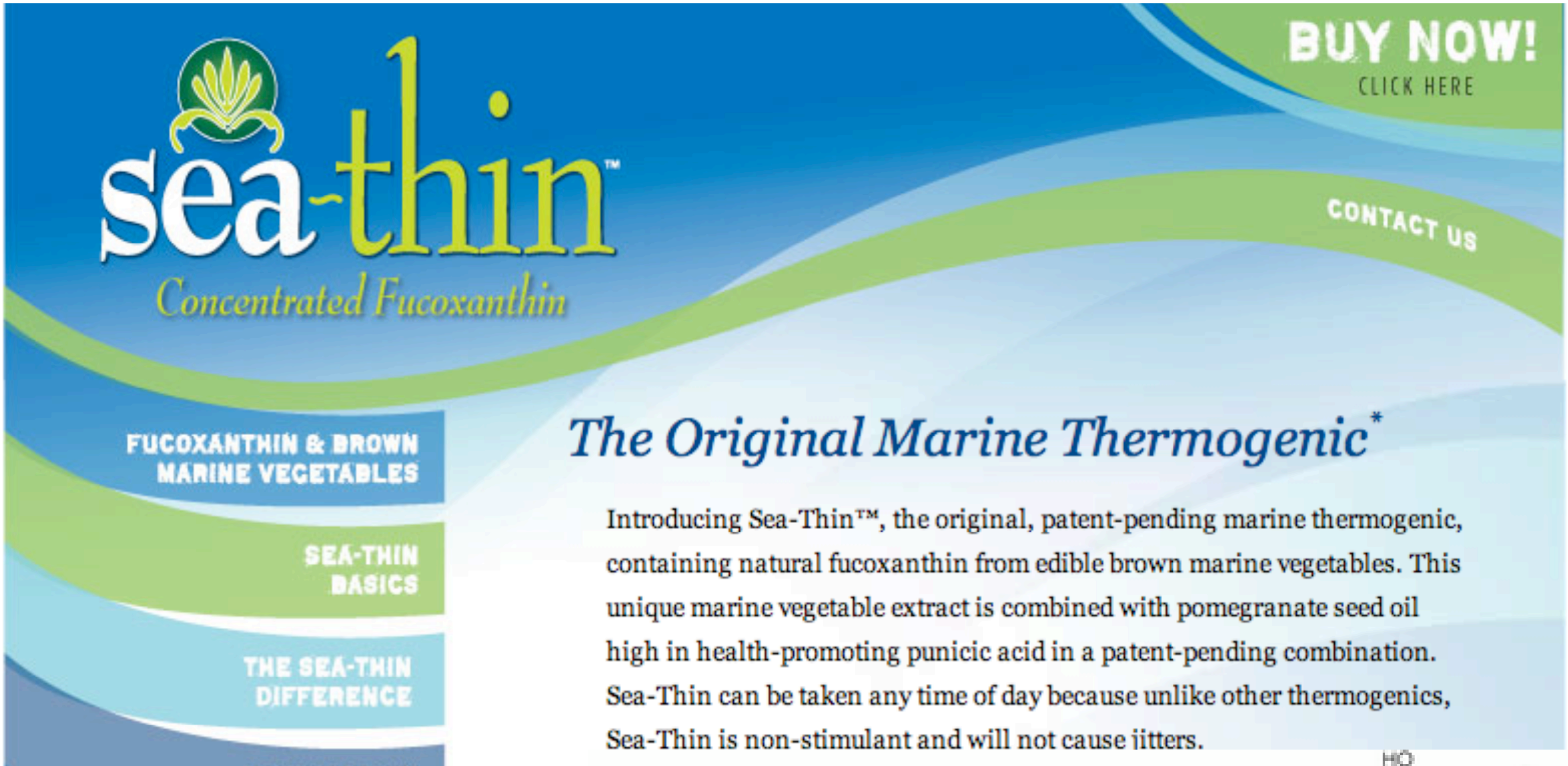
Harper JA, Dickinson K, Brand MD.

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

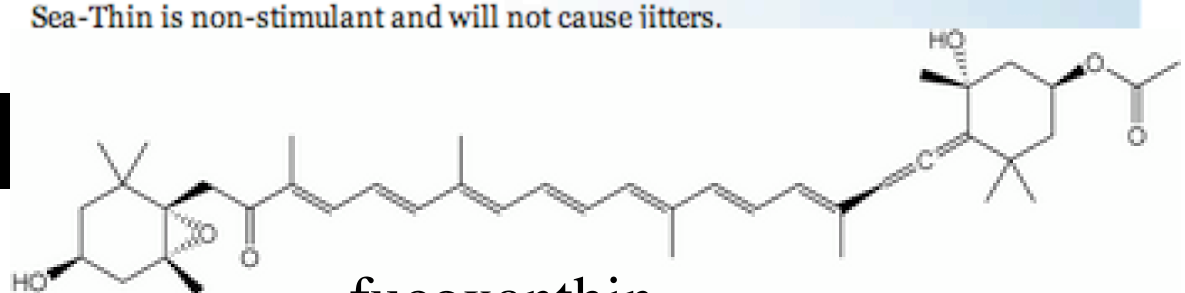
Mitochondrial proton cycling is responsible for a significant proportion of basal or standard metabolic rate, so further uncoupling of mitochondria may be a good way to increase energy expenditure and represents a good pharmacological target for the treatment of obesity. Uncoupling by 2,4-dinitrophenol 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 acyl 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-adrenoceptor 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?

Journal of Oleo Science

J. Oleo Sci. **56**, (12) 615-621 (2007)

JOURNAL OF OLEO SCIENCE
JOS

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

Hayato Maeda[1*], Masashi Hosokawa[1], Tokutake Sashima[2], Katsura Funayama[3] and Kazuo Miyashita[1]

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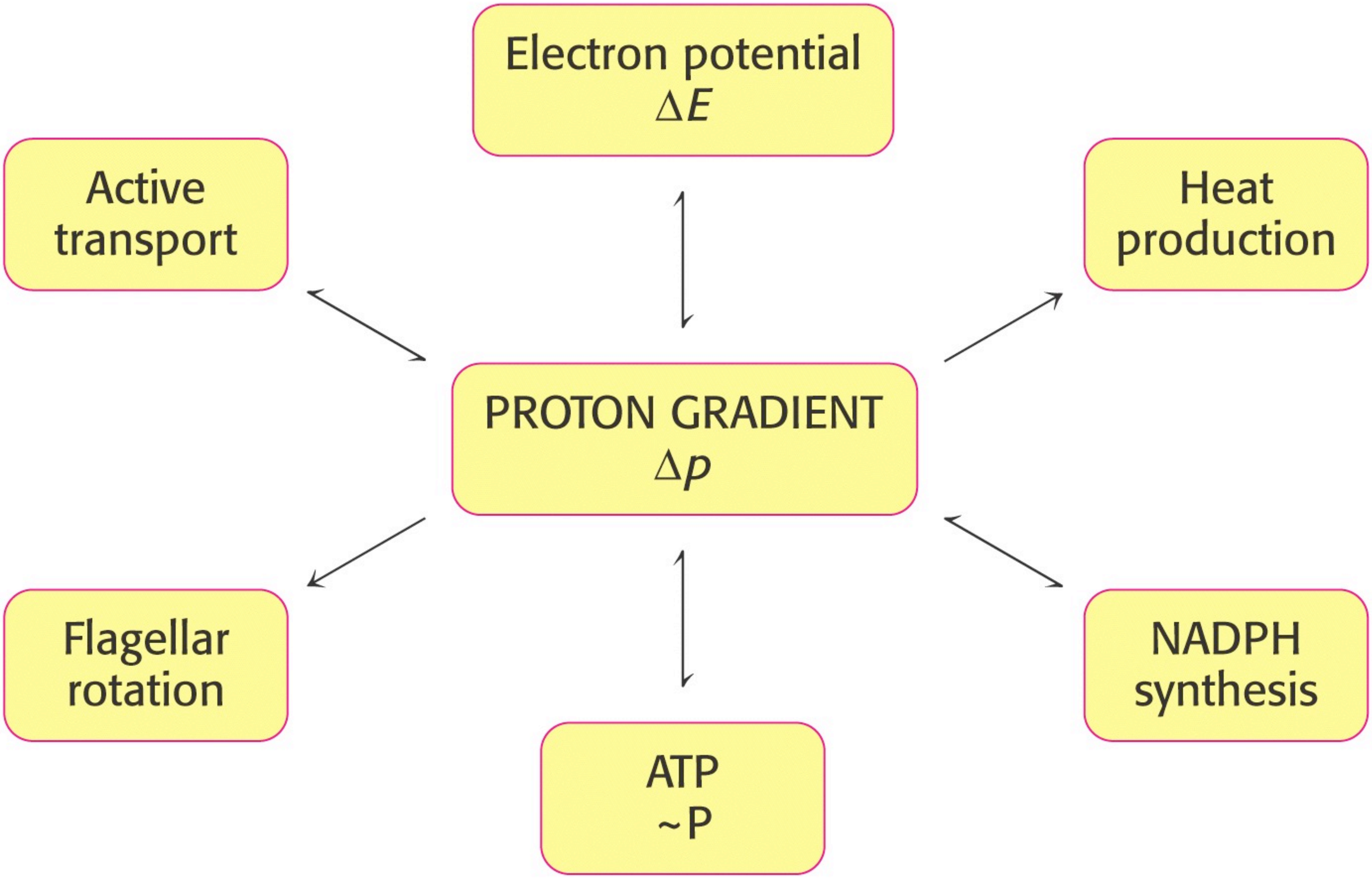
[2] Creative Research Institute, Hokkaido University (Hakodate, Hokkaido 041-8611, JAPAN)
[3] Riken Vitamin Co., (Tokyo 174-0065, JAPAN)

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.

words: fucoxanthin, obesity, medium-chain triacylglycerol

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


Electron potential
ΔE
Active
transport
Heat
production
PROTON GRADIENT
Δp
Flagellar
rotation
NADPH
synthesis
ATP
~P