# Chapter 19

*The Tricarboxylic Acid Cycle*

Biochemistry

by

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## *Essential Question*

- How is pyruvate oxidized under aerobic conditions

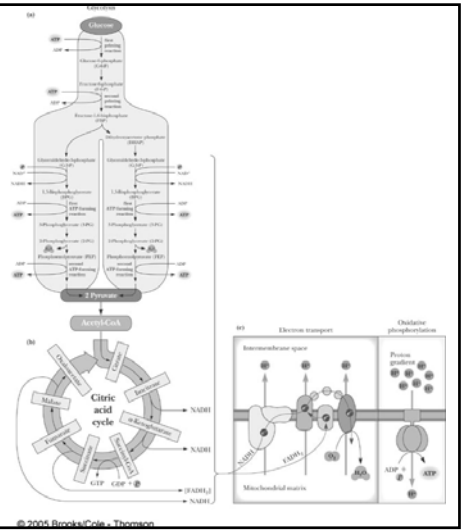
  Under aerobic conditions, pyruvate is converted to acetyl-CoA and oxidized to $CO_2$ in the TCA cycle

- What is the chemical logic that dictates how this process occurs?

## *Outline of chapter 19*

1. How Did Hans Krebs Elucidate the TCA Cycle?
2. What Is the Chemical Logic of the TCA Cycle?
3. How Is Pyruvate Oxidatively Decarboxylated to Acetyl-CoA?
4. How Are Two $CO_2$ Molecules Produced from Acetyl-CoA?
5. How Is Oxaloacetate Regenerated to Complete the TCA Cycle?
6. What Are the Energetic Consequences of the TCA Cycle?
7. Can the TCA Cycle Provide Intermediates for Biosynthesis?
8. What Are the Anaplerotic, or "Filling Up," Reactions?
9. How Is the TCA Cycle Regulated?
10. Can Any Organisms Use Acetate as Their Sole Carbon Source?

Figure 19.1
**(a)** Pyruvate produced in glycolysis is oxidized in **(b)** the tricarboxylic acid (TCA) cycle. **(c)** Electrons liberated in this oxidation flow through the electron-transport chain and drive the synthesis of ATP in oxidative phosphorylation. In eukaryotic cells, this overall process occurs in mitochondria.



*19.1 – How Did Hans Krebs Elucidate the TCA Cycle?*

*Citric Acid Cycle* or *Krebs Cycle*

- Pyruvate (actually acetate) from glycolysis is degraded to $CO_2$
- Some ATP is produced
- More NADH and $FADH_2$ are made
- NADH goes on to make more ATP in electron transport and oxidative phosphorylation (chapter20)

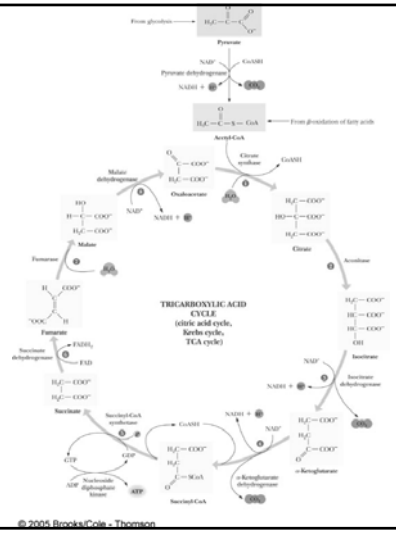
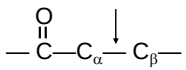


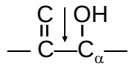

Figure 19.4
The tricarboxylic acid cycle.

*19.2 – What Is the Chemical Logic of the TCA Cycle?*

- TCA seems like a complicated way to oxidize acetate units to $CO_2$
- But normal ways to cleave C-C bonds and oxidize don't work for acetyl-CoA:
  1. cleavage between Carbons α and β to a carbonyl group



  2. α-cleavage of an α-hydroxyketone



*The Chemical Logic of TCA*
*A better way to cleave acetate...*

- Better to condense acetate with oxaloacetate and carry out a β-cleavage.
- TCA combines this β-cleavage reaction with oxidation to form $CO_2$, regenerate oxaloacetate and capture all the energy in NADH and ATP

## *19.3 – How Is Pyruvate Oxidatively Decarboxylated to Acetyl-CoA?*

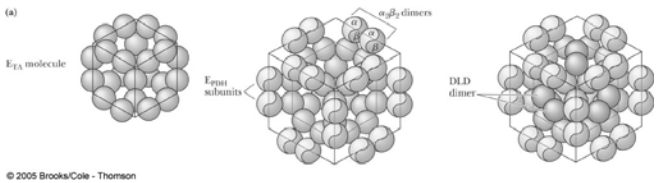
- Pyruvate must enter the mitochondria to enter the TCA cycle
- Oxidative decarboxylation of pyruvate is catalyzed by the pyruvate dehyrogenase complex

Pyruvate + CoA + $NAD^+$ $\longrightarrow$ acetyl-CoA + $CO_2$ + NADH + $H^+$

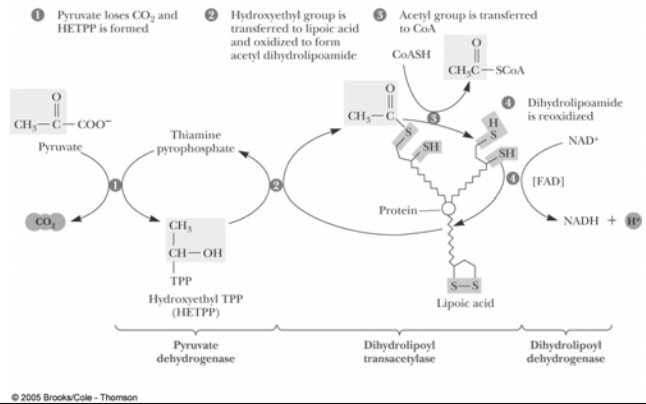
- Pyruvate dehydrogenase complex is a noncovalent assembly of three enzymes
- Five coenzymes are required

## *Pyruvate dehydrogenase complex:*

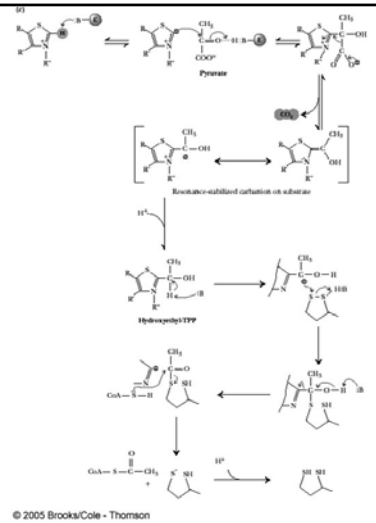
Three enzymes and five coenzymes
E1: pyruvate dehydrogenase (24)
thiamine pyrophosphate
E2: dihydrolipoyl transacetylase (24)
lipoic acid
E3: dihydrolipoyl dehydrogenase (12)
FAD
$NAD^+$
CoA




**(a)** The structure of the pyruvate dehydrogenase complex. This complex consists of three enzymes: **pyruvate dehydrogenase (PDH)**, **dihydrolipoyl transacetylase (TA)**, and **dihydrolipoyl dehydrogenase (DLD)**. (i) 24 dihydrolipoyl transacetylase subunits form a cubic core structure. (ii) 24 $\alpha\beta$ dimers of pyruvate dehydrogenase are added to the cube (two per edge). (iii) Addition of 12 dihydrolipoyl dehydrogenase subunits (two per face) completes the complex.




**(b)** The reaction mechanism of the pyruvate dehydrogenase complex. Decarboxylation of pyruvate occurs with formation of **hydroxyethyl-TPP** (Step 1). Transfer of the two-carbon unit to lipoic acid in Step 2 is followed by formation of acetyl-CoA in Step 3. Lipoic acid is reoxidized in Step 4 of the reaction.




**(c)** The mechanistic details of the first three steps of the pyruvate dehydrogenase complex reaction.

## *19.4 – How Are Two $CO_2$ Molecules Produced from Acetyl-CoA?*

### *Tricarboxylic acid cycle, Citric acid cycle, and Krebs cycle*

- Pyruvate is oxidatively decarboxylated to form acetyl-CoA

Citrate (6C)→ Isocitrate (6C)→ α-Ketoglutarate (5C) → Succinyl-CoA (4C) → Succinate (4C) → Fumarate (4C) → Malate (4C) → Oxaloacetate (4C)

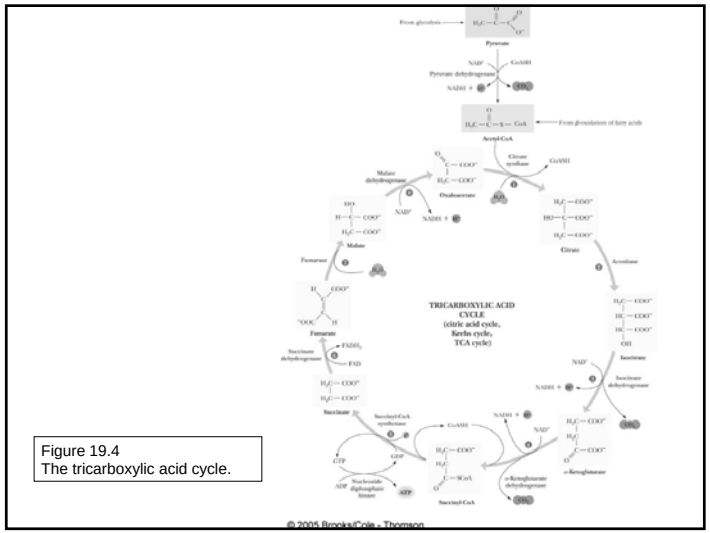



Figure 19.4
The tricarboxylic acid cycle.

## *Citrate synthase reaction*

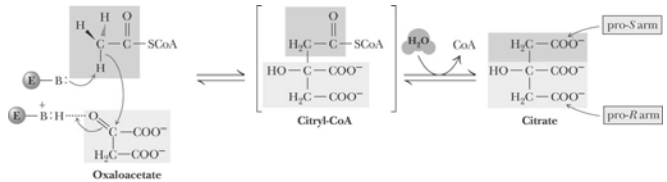



Figure 19.5
Citrate is formed in the citrate synthase reaction from oxaloacetate and acetyl-CoA. The mechanism involves nucleophilic attack by the carbanion of acetyl-CoA jon the carbonyl carbon of oxaloacetate, followed by thioester hydrolysis.

- Perkin condensation: a carbon-carbon condensation between a ketone or aldehyde and an ester

# *Citrate synthase reaction*

- Citrate synthase
  - is a dimer
  - NADH & succinyl-CoA are allosteric inhibitors
- Large, negative $\Delta G$ -- irreversible

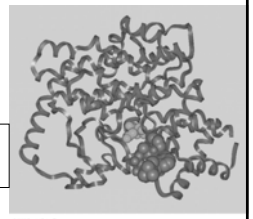


Figure 19.6
Citrate synthase. In the monomer shown here, citrate is shown in green, and CoA is pink.

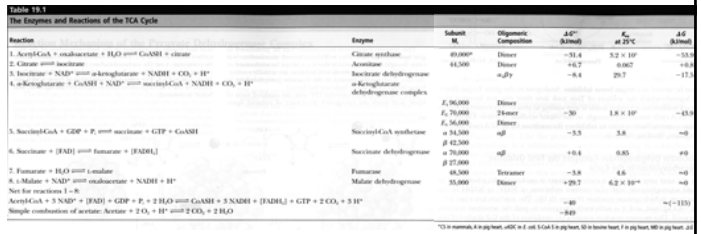


Table 19.1
The Enzymes and Reactions of the TCA Cycle

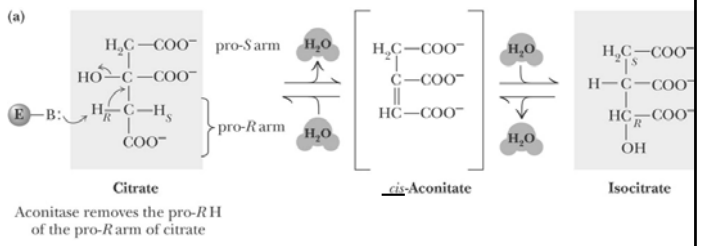
| Reaction | Enzyme | Subunit M$_r$ | Oligomeric Composition | $\Delta G^{\circ\prime}$ (kJ/mol) | $K_{eq}$ at 25°C | $\Delta G$ (kJ/mol) |
|---|---|---|---|---|---|---|
| 1. Acetyl-CoA + oxaloacetate + $H_2O$ ⟺ CoASH + citrate | Citrate synthase | 49,000* | Dimer | −31.4 | $5.2 \times 10^5$ | −55.9 |
| 2. Citrate ⟺ isocitrate | Aconitase | 61,500 | Dimer | +6.7 | 0.067 | +0.8 |
| 3. Isocitrate + $NAD^+$ ⟺ α-ketoglutarate + NADH + $CO_2$ + $H^+$ | Isocitrate dehydrogenase | | $\alpha_2\beta\gamma$ | −8.4 | 29.7 | −17.5 |
| 4. α-Ketoglutarate + CoASH + $NAD^+$ ⟺ succinyl-CoA + NADH + $CO_2$ + $H^+$ | α-Ketoglutarate dehydrogenase complex | | | | | |
| | | $E_1$ 96,000 | Dimer | | | |
| | | $E_2$ 70,000 | 24-mer | −30 | $1.8 \times 10^5$ | −43.9 |
| | | $E_3$ 56,000 | Dimer | | | |
| 5. Succinyl-CoA + GDP + P$_i$ ⟺ succinate + GTP + CoASH | Succinyl-CoA synthetase | α 34,500 | αβ | −3.3 | 3.8 | ≈0 |
| | | β 42,500 | | | | |
| 6. Succinate + [FAD] ⟺ fumarate + [$FADH_2$] | Succinate dehydrogenase | α 70,000 | αβ | +0.4 | 0.85 | ≈0 |
| | | β 27,000 | | | | |
| 7. Fumarate + $H_2O$ ⟺ L-malate | Fumarase | 48,500 | Tetramer | −3.8 | 4.6 | ≈0 |
| 8. L-Malate + $NAD^+$ ⟺ oxaloacetate + NADH + $H^+$ | Malate dehydrogenase | 35,000 | Dimer | +29.7 | $6.2 \times 10^{-6}$ | ≈0 |
| Net for reactions 1–8: | | | | | | |
| Acetyl-CoA + 3 $NAD^+$ + [FAD] + GDP + P$_i$ + 2 $H_2O$ ⟺ CoASH + 3 NADH + [$FADH_2$] + GTP + 2 $CO_2$ + 3 $H^+$ | | | | −40 | | ≈(−115) |
| Simple combustion of acetate: Acetate + 2 $O_2$ + $H^+$ ⟺ 2 $CO_2$ + 2 $H_2O$ | | | | −849 | | |

*C5 in mammals, A in pig heart, αEDC in E. coli, S CoA-S in pig heart, SD in bovine heart, F in pig heart, MD in pig heart. ΔG

# *Citrate Is Isomerized by Aconitase to Form Isocitrate*

## *Isomerization of Citrate to Isocitrate*

- Citrate is a poor substrate for oxidation
- So aconitase isomerizes citrate to yield isocitrate which has a secondary -OH, which can be oxidized
- Note the stereochemistry of the reaction: aconitase removes the pro-R H of the pro-R arm of citrate
- Aconitase uses an iron-sulfur cluster ( Fig. 19.8)



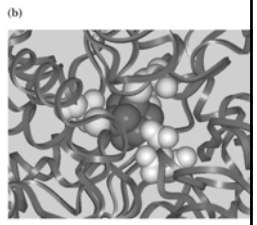
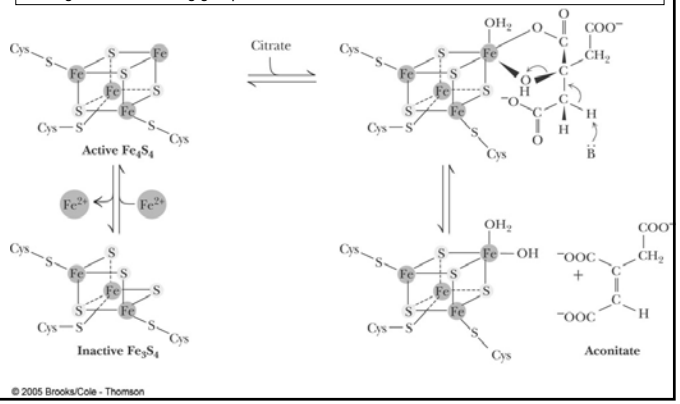




Figure 19.7
**(a)** The **aconitase reaction** converts citrate to *cis*-aconitate and then to isocitrate. Aconitase is stereospecific and removes the pro-*R* hydrogen from the pro-R arm of citrate. **(b)** The active site of aconitase. The **iron-sulfur cluster** (red) is coordinated by **cysteines (yellow)** and isocitrate (white).

Figure 19.8
The iron–sulfur cluster of aconitase. Binding of $Fe^{2+}$ to the vacant position of the cluster activates aconitase. The added iron atom coordinates the C-3 carboxyl and hydroxyl groups of citrate and acts as a Lewis acid, accepting an electron pair from the hydroxyl group and making it a better leaving group.






- Fluoroacetate is an extremely poisonous agent that blocks the TCA cycle
- Rodent poison: $LD_{50}$ is 0.2 mg/kg body weight
- Aconitase inhibitor

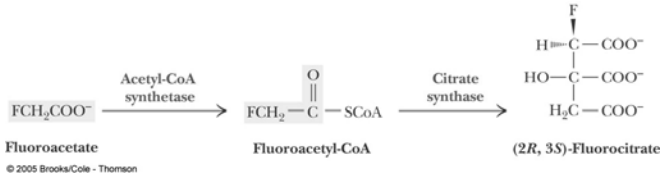





Figure 19.9
The conversion of fluoroacetate to fluorocitrate.

## *Isocitrate Dehydrogenase*

### *Oxidative decarboxylation of isocitrate to yield α-ketoglutarate*

- Catalyzes the first oxidative decarboxylation in the cycle
  1. Oxidation of C-2 alcohol of isocitrate with concomitant reduction of $NAD^+$ to NADH
  2. followed by a β-decarboxylation reaction that expels the central carboxyl group as $CO_2$
- Isocitrate dehydrogenase is a link to the electron transport pathway because it makes NADH
- α-ketoglutarate is also a crucial α-keto acid for aminotransferase reactions (Chapter 25)

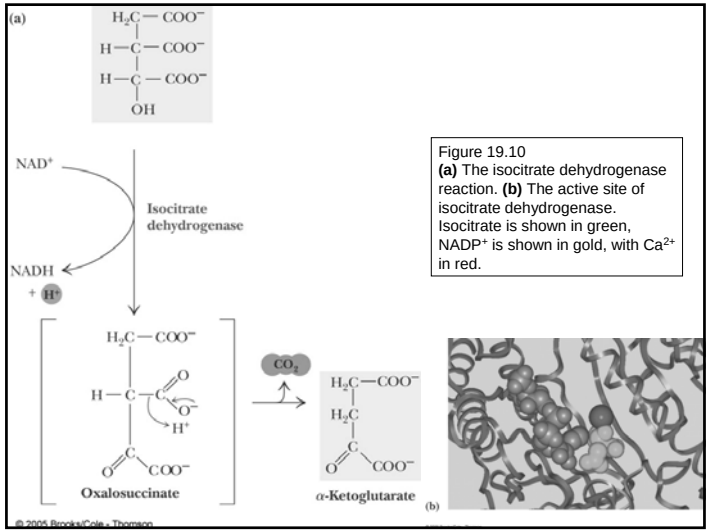





Figure 19.10
**(a)** The isocitrate dehydrogenase reaction. **(b)** The active site of isocitrate dehydrogenase. Isocitrate is shown in green, $NADP^+$ is shown in gold, with $Ca^{2+}$ in red.

## *α-Ketoglutarate Dehydrogenase*

- Catalyzes the second oxidative decarboxylation of the TCA cycle
- This enzyme is nearly identical to pyruvate dehydrogenase - structurally and mechanistically
  1. α-ketoglutarate dehydrogenase
  2. Dihydrolipoyl transsuccinylase
  3. Dihydrolipoyl dehydrogenase (identical to PDC)
- Five coenzymes used - TPP, CoA-SH, Lipoic acid, $NAD^+$, FAD

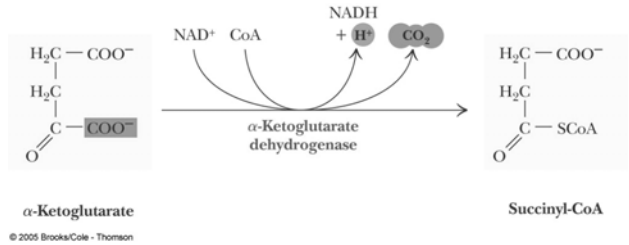



Figure 19.11
The $\alpha$-ketoglutarate dehydrogenase reaction.

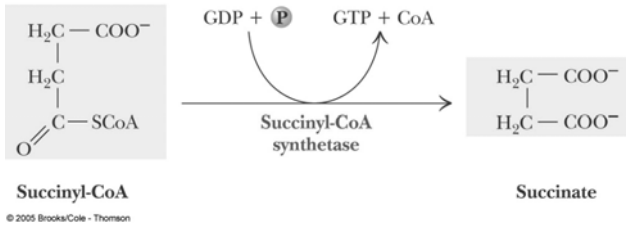
**Table 19.2**
**Composition of the α-Ketoglutarate Dehydrogenase Complex from *Escherichia coli***

| Enzyme | Coenzyme | Enzyme $M_r$ | Number of Subunits | Subunit $M_r$ | Number of Subunits per Complex |
|---|---|---|---|---|---|
| α-Ketoglutarate dehydrogenase | Thiamine pyrophosphate | 192,000 | 2 | 96,000 | 24 |
| Dihydrolipoyl transsuccinylase | Lipoic acid, CoASH | 1,700,000 | 24 | 70,000 | 24 |
| Dihydrolipoyl dehydrogenase | FAD, $NAD^+$ | 112,000 | 2 | 56,000 | 12 |

## *19.5 – How Is Oxaloacetate Regenerated to Complete the TCA Cycle?*

### *Succinyl-CoA Synthetase*

A substrate-level phosphorylation




- A nucleoside triphosphate is made

  GTP + ADP ↔ ATP + GDP

  (nucleotide diphosphate kinase)
- Its synthesis is driven by hydrolysis of a CoA ester
- The mechanism involves a phosphohistidine

Thioester

[Succinyl-P]

[Phospho-histidine]

GTP

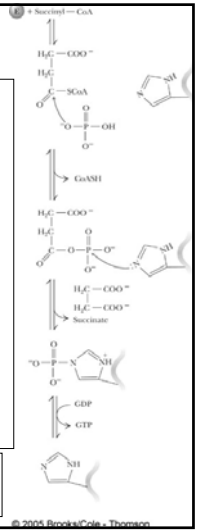



Figure 19.13
The mechanism of the succinyl-CoA synthetase reaction.

## *Succinate Dehydrogenase*

The oxidation of succinate to fumarate

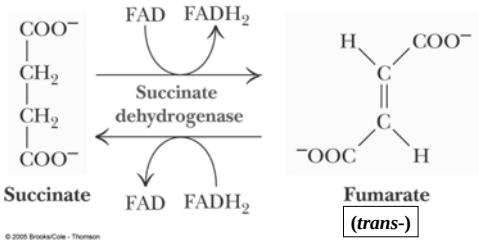



Figure 19.14 The succinate dehydrogenase reaction. Oxidation of succinate occurs with reduction of [FAD]. Reoxidation of [$FADH_2$] transfers electrons to coenzyme Q.

## *Succinate Dehydrogenase*

- A membrane-bound enzyme that is actually part of the electron transport chain in the inner mitochondrial membrane
- The electrons transferred from succinate to FAD (to form $FADH_2$) are passed directly to ubiquinone (UQ) in the electron transport pathway
- FAD is covalently bound to the enzyme

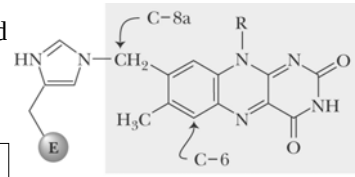



Figure 19.15 The covalent bond between FAD and succinate dehydrogenase involves the **C-8a** methylene group of FAD and the **N-3** of a histidine residue on the enzyme.

## *Succinate Dehydrogenase*

- Succinate oxidation involves removal of H atoms across a C-C bond, rather than a C-O or C-N bond
- The reaction is not sufficiently exergonic to reduce $NAD^+$
- Contains iron-sulfur cluster

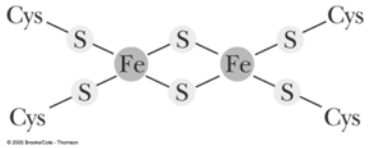



Figure 19.16
The $Fe_2S_2$ cluster of succinate dehydrogenase.

## *Fumarase*

*Hydration across the double bond*

- Catalyzes the *trans*-hydration of fumarate to form L-malate
- *trans*-addition of the elements of water across the double bond

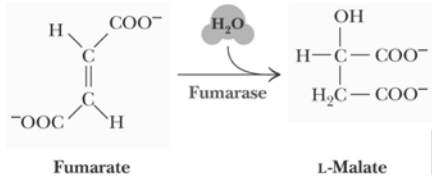
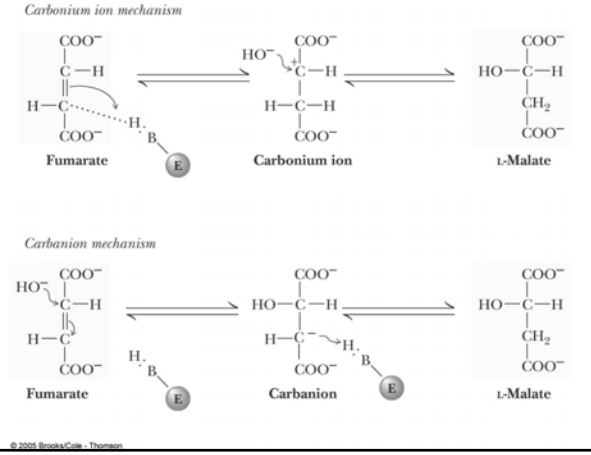



Figure 19.17
The fumarase reaction.

- Possible mechanisms are shown in Figure 19.18




## *Malate Dehydrogenase*

### *An NAD⁺-dependent oxidation*

- Completes the Cycle by Oxidizing Malate to Oxaloacetate
- The carbon that gets oxidized is the one that received the -OH in the previous reaction
- This reaction is very endergonic, with a $\Delta G^{o'}$ of +30 kJ/mol
- The concentration of oxaloacetate in the mitochondrial matrix is quite low

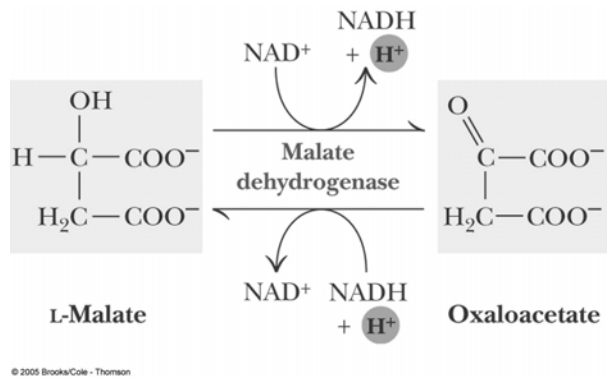
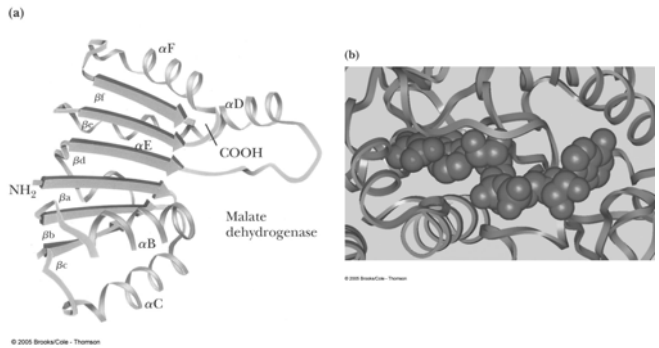
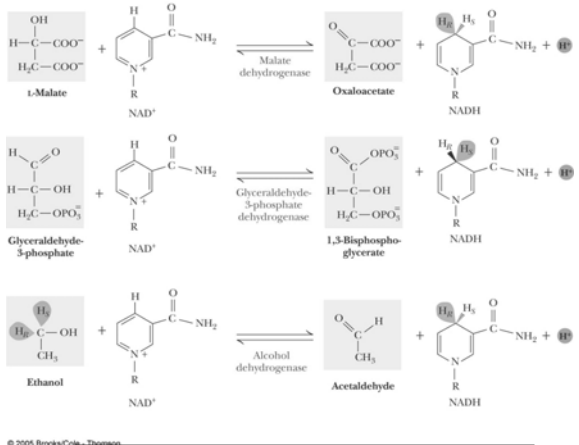



Figure 19.19
The malate dehydrogenase reaction.

Figure 19.20
**(a)** The structure of malate dehydrogenase. **(b)** The active site of malate dehydrogenase. **Malate is shown in red**; **NAD⁺ is blue**.

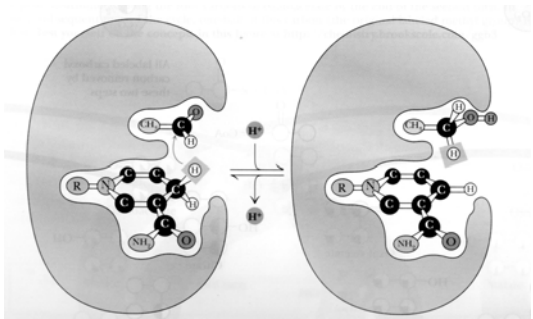





A Deeper Look
Steric Preferences in $NAD^+$ Dependent Dehydrogenases

**Steric Specificity for NAD of Various Pyridine Nucleotide-Linked Enzymes**

| Dehydrogenase | Source | Steric Specificity |
|---|---|---|
| Alcohol (with ethanol) | Yeast, *Pseudomonas*, liver, wheat germ | $H_R$ |
| Alcohol (with isopropyl alcohol) | Yeast | |
| Acetaldehyde | Liver | |
| L-Lactate | Heart muscle, *Lactobacillus* | |
| L-Malate | Pig heart, wheat germ | |
| D-Glycerate | Spinach | |
| Dihydroorotate | *Zymobacterium oroticum* | |
| α-Glycerophosphate | Muscle | $H_S$ |
| Glyceraldehyde-3-P | Yeast, muscle | |
| L-Glutamate | Liver | |
| D-Glucose | Liver | |
| β-Hydroxysteroid | *Pseudomonas* | |
| NADH cytochrome *c* reductase | Rat liver mitochondria, pig heart | |
| NADPH transhydrogenase | *Pseudomonas* | |
| NADH diaphorase | Pig heart | |
| L-β-Hydroxybutyryl-CoA | Heart muscle | |



## *19.6 – What Are the Energetic Consequences of the TCA Cycle?*

*One acetate through the cycle produces two $CO_2$, one ATP, four reduced coenzymes*

Acetyl-CoA + 3 $NAD^+$ + FAD + ADP + Pi + 2 $H_2O$ →
2 $CO_2$ + 3 NADH + 3 $H^+$ + $FADH_2$ + ATP +CoASH
$\Delta G^{0'}$ = -40kJ/mol

Glucose + 10 $NAD^+$ + 2 FAD + 4 ADP + 4 Pi + 2 $H_2O$ →
6 $CO_2$ + 10 NADH + 10 $H^+$ + 2 $FADH_2$ + 4 ATP

NADH + $H^+$ + 1/2 $O_2$ + 3 ADP + 3 Pi → $NAD^+$ + 3ATP + $H_2O$
$FADH_2$ + 1/2 $O_2$ + 2 ADP + 2 Pi → FAD + 2ATP + $H_2O$

## *The Carbon Atoms of Acetyl-CoA Have Different Fates in the TCA Cycle*

- Neither of the carbon atoms of a labeled acetate unit is lost as $CO_2$ in the first turn of the cycle
- Carbonyl C of acetyl-CoA turns to $CO_2$ only in the second turn of the cycle (following entry of acetyl-CoA )
- Methyl C of acetyl-CoA survives two cycles completely, but half of what's left exits the cycle on each turn after that.

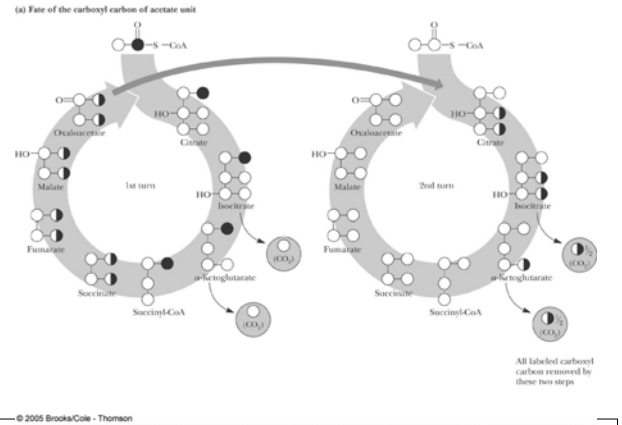



Figure 19.21
The fate of the carbon atoms of acetate in successive TCA cycles. **(a)** The carbonyl carbon of acetyl-CoA is fully retained through one turn of the cycle but is lost completely in a second turn of the cycle.

## *The Carbon Atoms of Acetyl-CoA Have Different Fates in the TCA Cycle*

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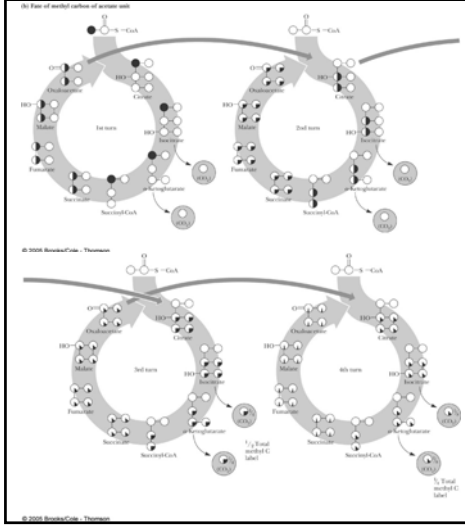


Figure 19.21
**(b)** The methyl carbon of a labeled acetyl-CoA survives two full turns of the cycle but becomes equally distributed among the four carbons of oxaloacetate by the end of the second turn. In each subsequent turn of the cycle, one-half of this carbon (the original labeled methyl group) is lost.

*The Carbon Atoms of Oxaloaceate in the TCA Cycle*

- Both of the carbonyl carbons of oxaloaceate are lost as $CO_2$, but the methylene and carbonyl carbons survive through the second turn
- The methylene carbon survives two full turns of cycle
- The carbonyl carbon is the same as the methyl carbon of acetyl-CoA

*19.7 – Can the TCA Cycle Provide Intermediates for Biosynthesis?*

The products in TCA cycle also fuel a variety of biosynthetic processes

- $\alpha$-Ketoglutarate is transaminated to make glutamate, which can be used to make purine nucleotides, Arg and Pro
- Succinyl-CoA can be used to make porphyrins
- Fumarate and oxaloacetate can be used to make several amino acids and also pyrimidine nucleotides
- Oxaloacetate can also be decarboxylated to yield PEP

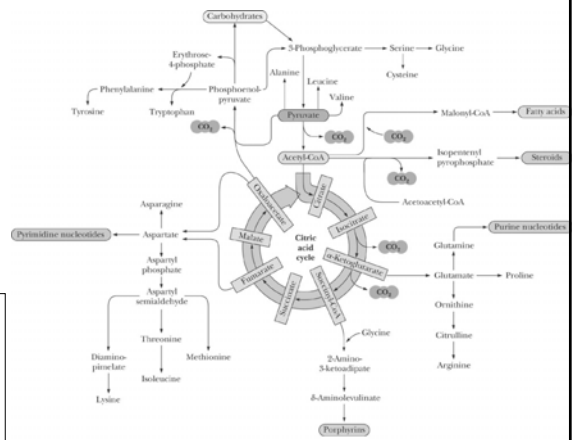



Figure 19.22 The TCA cycle provides intermediates for numerous biosynthetic processes in the cell.

*Intermediates for Biosynthesis*
*The TCA cycle provides several of these*

- Citrate can be exported from the mitochondria and then broken down by citric lyase to yield acetyl-CoA and oxaloacetate
- Oxaloacetate is rapidly reduced to malate
- Malate can be transported into mitochondria or oxidatively decarboxylated to pyruvate by malic enzyme

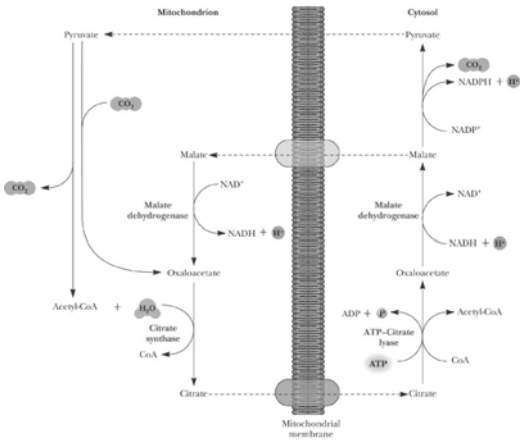



Figure 19.23
Export of citrate from mitochondria and cytosolic breakdown produces oxaloacetate and acetyl-CoA. Oxaloacetate is recycled to malate or pyruvate, which reenters the mitochondria. This cycle provides acetyl-CoA for fatty acid synthesis in the cytosol.

## *19.8 – What Are the Anaplerotic, or "Filling Up," Reactions?*

- PEP carboxylase - converts PEP to oxaloacetate (in bacteria & plants), inhibited by aspartate
- Pyruvate carboxylase - converts pyruvate to oxaloacetate (in animals), is activated by acetyl-CoA
- Malic enzyme converts pyruvate into malate
- PEP carboxykinase - could have been an anaplerotic reaction. $CO_2$ binds weakly to the enzyme, but oxaloacetate binds tightly, so the reaction favors formation of PEP from oxaloacetate

Figure 19.24
Phosphoenolpyruvate (PEP) carboxylase, pyruvate carboxylase, and malic enzyme catalyze anaplerotic reactions, replenishing TCA cycle intermediates.

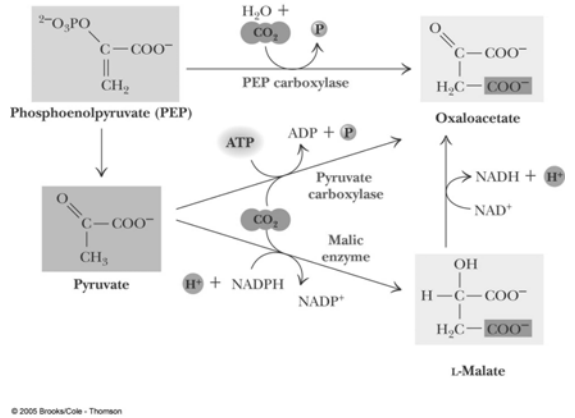
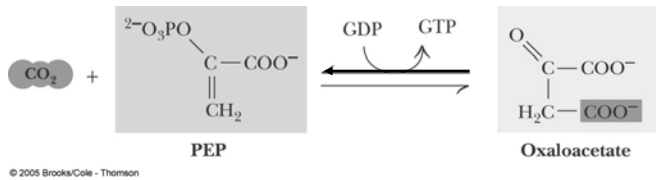



Figure 19.25
The phosphoenolpyruvate carboxykinase reaction.



## *19.9 – How Is the TCA Cycle Regulated?*

- Citrate synthase - ATP, NADH and succinyl-CoA inhibit
- Isocitrate dehydrogenase - ATP and NADH inhibits, ADP and $NAD^+$ activate
- $\alpha$ -Ketoglutarate dehydrogenase - NADH and succinyl-CoA inhibit, AMP activates
- Also note pyruvate dehydrogenase: ATP, NADH, acetyl-CoA inhibit, $NAD^+$, CoA activate
- When the ADP/ATP or $NAD^+$/NADH ratio is high, the TCA cycle is turned on

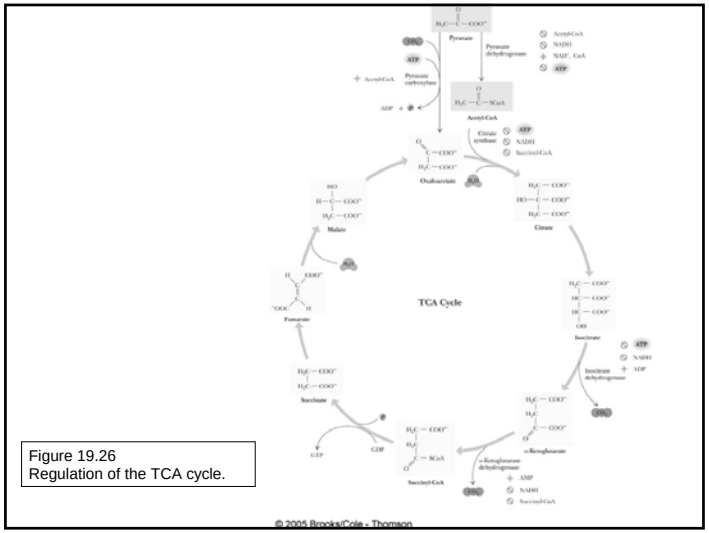



Figure 19.26
Regulation of the TCA cycle.

## Pyruvate dehydrogenase is regulated by phosphorylation/dephosphorylation

- Animals cannot synthesize glucose from acetyl-CoA, so pyruvate dehydrogenase is carefully regulated enzyme
- Acetyl-CoA (dihydrolipoyl transacetylase), or NADH (dihydrolipoyl dehydrogenase) allosterically inhibit
- Is also regulated by covalently modification, phosphorylation (pyruvate dehydrogenase kinase) and dephosphorylation (pyruvate dehydrogenase phosphatase) on pyruvate dehydrogenase

## Pyruvate dehydrogenase is regulated by phosphorylation/dephosphorylation

- The pyruvate dehydrogenase kinase is associated with the enzyme and allosterically activated by NADH and acetyl-CoA
- Phosphorylated pyruvate dehydrogenase subunit is inactive
- Reactivation of the enzyme by pyruvate dehydrogenase phosphatase, a $Ca_2^+$-activated enzyme

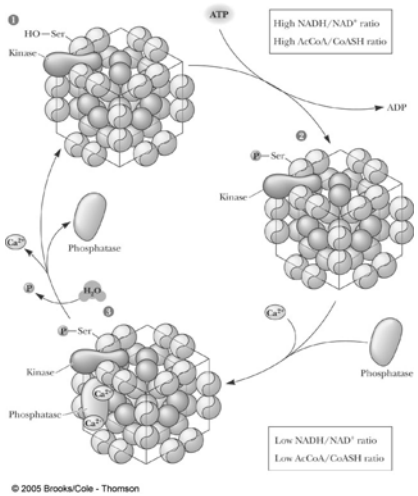



Figure 19.27
Regulation of the pyruvate dehydrogenase reaction.

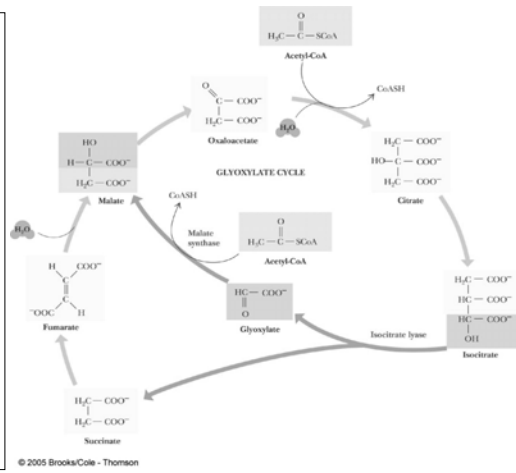
- At low ratios of NADH/$NAD^+$ and low acetyl-CoA levels, the phosphatase maintains the dehydrogenase in an activated state
- A high level of acetyl-CoA or NADH once again activates the kinase
- Insulin and $Ca_2^+$ ions activate dephosphorylation
- Pyruvate inhibits the phosphorylation reaction

## *19.10 – Can Any Organisms Use Acetate as Their Sole Carbon Source?*

### *The Glyoxylate Cycle*

- Plant can use acetate as the only source of carbon for all the carbon compounds
- Glyoxylate cycle offers a solution for plants and some bacteria and algae
- The $CO_2$-producing steps are bypassed and an extra acetate is utilized
- Isocitrate lyase and malate synthase are the short-circuiting enzymes

Figure 19.28
The glyoxylate cycle. The first two steps are identical to TCA cycle reactions. The third step bypasses the $CO_2$-evolving steps of the TCA cycle to produce succinate and glyoxylate. The malate synthase reaction forms malate from glyoxylate and another acetyl-CoA. The result is that one turn of the cycle consumes one oxaloacetate and two acetyl-CoA molecules but produces two molecules of oxaloacetate. The net for this cycle is one oxaloacetate from two acetyl-CoA molecules.



## *Glyoxylate Cycle*

- Isocitrate lyase produces glyoxylate and succinate
- Malate synthase does a Claisen condensation of acetyl-CoA and the aldehyde group of glyoxylate to form L-malate
- In plants, the glyoxylate cycle is carried out in glyoxysomes, but yeast and algae carry out in cytoplasm

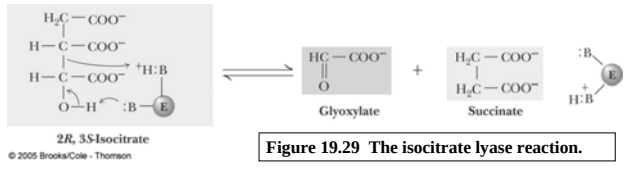



**Figure 19.29 The isocitrate lyase reaction.**

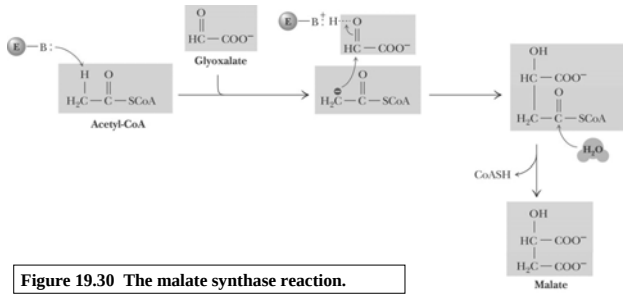



**Figure 19.30 The malate synthase reaction.**

## *Glyoxylate Cycle*

- The glyoxylate cycle helps plants grow in the dark
  - Once the growing plant begins photosynthesis and can fix CO2 to produce carbohydrate, the glyoxysomes disappear
- Glyoxysomes borrow three reactions from mitochondria: succinate to oxaloacetate
  1. Succinate dehydrogenase
  2. Fumarate
  3. Malate dehydrogenase

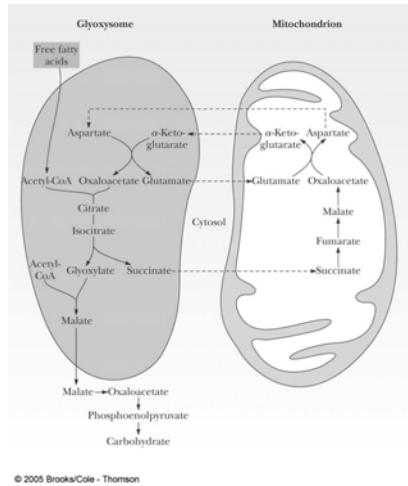



Figure 19.31 Glyoxysomes lack three of the enzymes needed to run the glyoxylate cycle. Succinate dehydrogenase, fumarase, and malate dehydrogenase are all "borrowed" from the mitochondria in a shuttle in which succinate and glutamate are passed to the mitochondria, and $\alpha$-ketoglutarate and aspartate are passed to the glyoxysome.