# Beta-oxidation

β-oxidation is a cyclic mechanism to utilize fatty acids. The product is a significant amount of Acetyl-CoA, which proceeds to the citrate cycle, and reduced coenzymes, which are used in the respiratory chain.

## General β-oxidation

Higher fatty acids are oxidized in the matrix of mitochondria. In addition to activation by coenzyme A, fatty acids from 12C length must use the carnitine transporter, which is located between the outer and inner membranes of the mitochondria. Acids longer than 18C must first be shortened at the endoplasmic reticulum [1] or peroxisome.

Oxidation of acids occurs at the β-carbon (i.e. C3), therefore the reaction is called β-oxidation. Its course is aerobic and dependent on the functionality of the respiratory chain.

The enzymes represented in the reactions are specific for short, medium and long chains. Medium-chain acyl-CoA dehydrogenase deficiency is associated with sudden infant death syndrome (SIDS) and Reye's syndrome. A total of four enzymes [2] are used in the reactions:

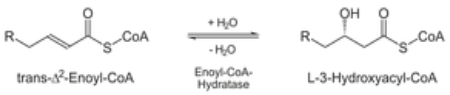
- **Acyl-CoA-dehydrogenase** - takes two hydrogens and passes them to FAD;
- **enoyl-CoA-hydratase** – ensures the addition of water to the double bond, resulting in a hydroxyl group;
- **β-hydroxyacyl-CoA-dehydrogenase** – takes two hydrogens by further dehydrogenation to form NADH+H + ;
- **thiolase** – cleaves the resulting oxoacyl to form Ac-CoA and attach HS-CoA to acyl. This acyl-CoA is now 2 carbons shorter and can rejoin the beginning of the cycle.

## Response overview

**1. Acyl-CoA + FAD → unsaturated acyl-CoA + $FADH_2$**

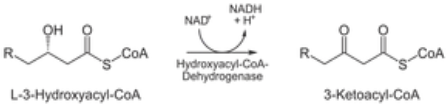

- Dehydrogenation and double bond formation occur at the β-carbon of acyl-CoA, reducing the flavin coenzyme.
- Catalyzed by the enzyme **acyl-CoA dehydrogenase**.

**2. Unsaturated acyl-CoA + H 2 O → β-hydroxyacyl-CoA**



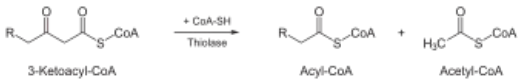

- Addition of water to the double bond.
- Catalyzed by the enzyme enoyl-CoA-hydratase .

**3. β-hydroxyacyl-CoA + NAD+ → β-oxoacyl-CoA + NADH + $H^+$**




- Dehydrogenation of the hydroxyl group and β-carbon to form NADH+$H^+$.
- Catalyzed by the enzyme **β-hydroxyacyl-CoA-dehydrogenase**.

**4. β-oxoacyl-CoA + HS-CoA → Ac-CoA + acyl-CoA(-2C)**




- The final step, when Acetyl-CoA is cleaved and the chain is thus shortened by two carbons.
- Catalyzed by the enzyme **thiolase**.

## Yield of β-oxidation[3]

Fatty acids are a rich source of both Ac-CoA and hydrogen, which reduce the respective FAD and NAD+ coenzymes. They are then used in the respiratory chain.

The approximate yield for palmitic acid (C16) is:

- 8x Ac-CoA = 80 ATP (2x Ac-CoA is formed from the four-carbon acyl-CoA in the last step);
- 7x NADH+$H^+$ = 17.5 ATP;
- 7x $FADH_2$ = 10.5 ATP;
- loss of 2 ATP for acyl activation.

In total, the theoretical yield is therefore 106 ATP.

## Regulation of β-oxidation

The cycle is affected by[4]:

- by the enzyme **carnitine transferase**, i.e. the carnitine carrier;
- the availability of **substrates (fatty acids, carnitine)**;
- **by pumping out products** ;
- **respiratory control**.

Hormonally:

- insulin ↓ (inhibits);
- glucagon ↑, adrenaline ↑ (activates).

# Modified β-oxidation[5]

In cases of degradation of **unsaturated fatty acids**, a modification of the cycle occurs. The vast majority have their double bonds in the cis position . Until the mechanism encounters this bond, β-oxidation proceeds in the classical manner. **The enzyme isomerase** then intervenes to convert the double bond from the cis position to the trans position. Next, oxidation continues again in the classic way.

Another change in the cycle is the degradation of fatty acids longer than 18 carbons. Here comes the peroxisome, whose role is to shorten the chains under 18C. These are then taken up by the mitochondria for processing.

The last and less significant case is the β-oxidation of fatty acids with an odd number of carbons. In the last stage of classical β-oxidation, **propionyl-CoA** remains , which is converted to succinyl-CoA. It serves as a substrate in the citrate cycle. It is also the only exception where a fatty acid can be a substrate of gluconeogenesis.

# Links

## Related Articles

- Citrate cycle]
- Respiratory chain
- Acetyl-CoA
- Carnitine transport system
- Regulation of individual metabolic pathways
- Ketone bodies

## References


- LEDVINA, Miroslav, et al. *Biochemistry for medical student. Part I.* 2. edition. 0000. pp. 157-162. ISBN 978-80-246-1416-8.
- DUŠKA, František. *Biochemistry for medical student. Part I.* 1. edition. Karolinum, 2006. pp. 75-79. ISBN 80-246-1116-3.

1. DUŠKA, František. *Biochemie v souvislostech, 1.díl – základy energetického metabolizmu.* 1. edition. Praha : Karolinum, 2006. 165 pp. pp. 77. ISBN 80-246-1116-3.
2. LEDVINA, Miroslav, et al. *Biochemie pro studující medicíny. I. díl.* 2. edition. Praha : Karolinum, 0000. 269 pp. pp. 159. ISBN 978-80-246-1416-8.
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4. LEDVINA, Miroslav, et al. *Biochemie pro studující medicíny. I. díl.* 2. edition. Praha : Karolinum, 0000. 269 pp. pp. 160. ISBN 978-80-246-1416-8.
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