# COX10 Polyclonal Antibody

**catalog number:** E-AB-13841

**Note:** *Centrifuge before opening to ensure complete recovery of vial contents.*

## Description

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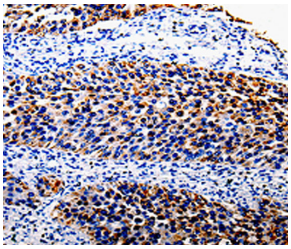
|---|---|
| **Reactivity** | Human |
| **Immunogen** | Recombinant protein of human COX10 |
| **Host** | Rabbit |
| **Isotype** | IgG |
| **Purification** | Affinity purification |
| **Conjugation** | Unconjugated |
| **Buffer** | Phosphate buffered solution, pH 7.4, containing 0.05% stabilizer and 50% glycerol. |

| Applications | Recommended Dilution |
|---|---|
| **IHC** | 1:50-1:200 |

## Data



Immunohistochemistry of paraffin-embedded Human renal cancer tissue using COX10 Polyclonal Antibody at dilution 1:100

## Preparation & Storage

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|---|---|
| **Storage** | Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles. |
| **Shipping** | The product is shipped with ice pack,upon receipt,store it immediately at the temperature recommended. |

## Background

Cytochrome c oxidase (COX), the terminal component of the mitochondrial respiratory chain, catalyzes the electron transfer from reduced cytochrome c to oxygen. This component is a heteromeric complex consisting of 3 catalytic subunits encoded by mitochondrial genes and multiple structural subunits encoded by nuclear genes. The mitochondrially-encoded subunits function in electron transfer, and the nuclear-encoded subunits may function in the regulation and assembly of the complex. This nuclear gene encodes heme A:farnesyltransferase, which is not a structural subunit but required for the expression of functional COX and functions in the maturation of the heme A prosthetic group of COX. This protein is predicted to contain 7-9 transmembrane domains localized in the mitochondrial inner membrane. A gene mutation, which results in the substitution of a lysine for an asparagine (N204K), is identified to be responsible for cytochrome c oxidase deficiency. In addition, this gene is disrupted in patients with CMT1A ( Charcot-Marie-Tooth type 1A) duplication and with HNPP (hereditary neuropathy with liability to pressure palsies) deletion.