

## Detection of exon 18 EGFR mutations -Sequencing-

**AMPLI-EGFR seq exon 18**

**Cat. n. 2.008 seq**

EGFR (epidermal growth factor receptor) is a membrane receptor tyrosine kinase belonging to the family of ErbB receptors. This receptor, once bound its specific ligand EGF (epidermal growth factor) and TGF $\alpha$  (transforming growth factor  $\alpha$ ), activates multiple signal transduction pathways that regulate various cellular processes: division, apoptosis, motility, adhesion.

EGFR mutations are implicated in about 30% of all epithelial tumors. These mutations are located in exons 4 (EX18-21) in the region of the ATP binding site of the tyrosine kinase domain. These mutations cause constitutive activation of the EGFR tyrosine portion, destabilizing its conformation self inhibitor, normally maintained in the absence of ligand, these activating mutations confer hypersensitivity to the inhibitors gefitinib and erlotinib tyrosine kinases. Several retrospective studies have shown that EGFR mutations are an independent predictor of response, overall survival (OS) and progression-free survival (PFS) in patients with metastatic non-small cell lung cancer (NSCLC) treated with gefitinib, the most of whom underwent prior chemotherapy.

The method involves amplification of the gene regions of interest by polymerase chain reaction (PCR) with primers specific. The amplification products can be subjected to sequencing using the primers provided by the kit.

**Principle of method:**

- a) isolation of genomic DNA
- b) amplification;
- c) sequencing;

**Applicability:** genomic DNA isolated and purified by whole blood, fresh or paraffin embedded tissues

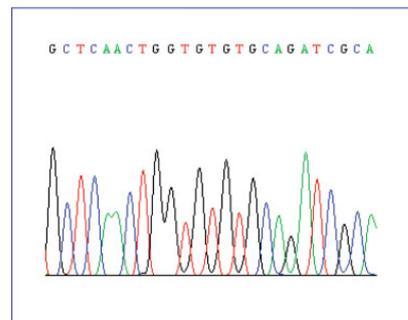
**Number of tests :** 24.

**Kit contains and storage**

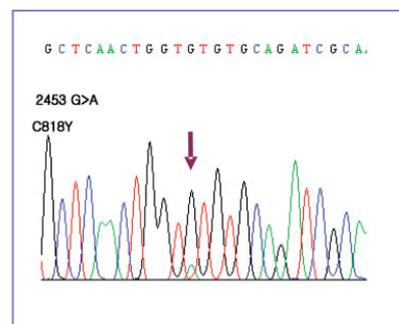
<b>AMPLIFICATION</b>	
Mix PCR ex 18	-20°C
H <sub>2</sub> O DNase/RNase-free	-20°C
Taq Polymerase (5U/ $\mu$ l)	-20°C
Sequencing Primer ex21 (100 pmoli/ $\mu$ l)	-20°C

**Stability:** more than 18 months if properly stored.

**ANALYSIS OF RESULTS**



Exon 18 wt



Exon 18 mut

**References**

Science (2004) 304, 1497-1500.  
 N Engl J Med (2004) 350, 2129-2139.  
 Proc Natl Acad Sci U S A (2004) 101, 13306-13311.  
 L Clin Oncol (2005), 23:2513-2520.