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# Product datasheet for AP14370PU-N

## Insulin Receptor (INSR) (N-term) Rabbit Polyclonal Antibody

### Product data:

| | |
|---|---|
| **Product Type:** | Primary Antibodies |
| **Applications:** | IHC, WB |
| **Recommended Dilution:** | ELISA: 1/1,000.<br>Western blotting: 1/100 - 1/500.<br>Immunohistochemistry: 1/50 - 1/100. |
| **Reactivity:** | Human |
| **Host:** | Rabbit |
| **Isotype:** | Ig |
| **Clonality:** | Polyclonal |
| **Immunogen:** | This antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide selected from the N-terminal region of human INSR. |
| **Specificity:** | This antibody reacts to INSR (Insulin Receptor). |
| **Formulation:** | PBS with 0.09% (W/V) sodium azide<br>State: Purified<br>State: Liquid purified Ig |
| **Concentration:** | lot specific |
| **Purification:** | Protein G column, eluted with high and low pH buffers and neutralized immediately, followed by dialysis against PBS |
| **Conjugation:** | Unconjugated |
| **Storage:** | Store the antibody undiluted at 2-8°C for one month or (in aliquots) at -20°C for longer.<br>Avoid repeated freezing and thawing. |
| **Stability:** | Shelf life: one year from despatch. |
| **Gene Name:** | insulin receptor |
| **Database Link:** | Entrez Gene 3643 Human<br>P06213 |

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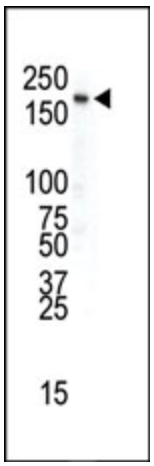
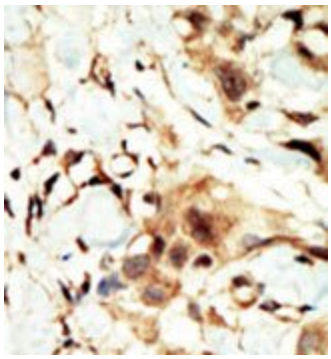
**Background:** INSR is a receptor that binds insulin and has a tyrosine-protein kinase activity. Autophosphorylation activates the kinase activity. This Type I mebrane protein is composed of a tetramer of 2 alpha and 2 beta chains linked by disulfide bonds. The alpha chains contribute to the formation of the ligand-binding domain, while the beta chains carry the kinase domain. After being transported from the endoplasmic reticulum to the Golgi apparatus, the single glycosylated precursor is further glycosylated and then cleaved, followed by its transport to the plasma membrane. Defects in INSR are the cause of insulin resistance of various forms, including mild insulin-resistant diabetes mellitus with acanthosis nigricans, minor physical abnormalities and sometimes polycystic ovaries. Insulin resistance associated with acanthosis nigricans, hirsutism and hyperandrogenism is referred to as insulin resistance type A. Defects in INSR are the cause of Rabson-Mendenhall syndrome, also known as Mendenhall syndrome. It is a severe insulin resistance syndrome characterized by insulin-resistant diabetes mellitus with pineal hyperplasia and somatic abnormalities. Typical features include coarse, senile-appearing facies, dental and skin abnormalities, abdominal distension, and phallic enlargement. Inheritance is autosomal recessive. Defects in INSR are the cause of leprechaunism, also known as Donohue syndrome. Leprechaunism represents the most severe form of insulin resistance syndrome, characterized by intrauterine and postnatal growth retardation and death in early infancy. Inheritance is autosomal recessive. Defects in INSR may be associated with noninsulin-dependent diabetes mellitus.

**Synonyms:** Insulin Receptor

## Product images:




Western blot analysis of anti-INSR Pab in SKBR-3 cell lysate. INSR (arrow) was detected using purified Pab. Secondary HRP-anti-rabbit was used for signal visualization with chemiluminescence.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by DAB staining.