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## **ATXN2 (M) Antibody, Rabbit Polyclonal**

**Cat#: R1561-2**

**Quantity: 100 ul**

**Predicted | Observed M.W.: 140 | 170 kDa**

**Lot#: Refer to vial**

**Application: WB**

**Uniprot ID: Q99700**

### **Background:**

Ataxin-2 (ATXN2) is involved in EGFR trafficking, acting as a negative regulator of the endocytic EGFR internalization at the plasma membrane. Defects in ATXN2 are the cause of spinocerebellar ataxia type 2 (SCA2). SCA2 belongs to the autosomal dominant cerebellar ataxias type I (ADCA I), which are characterized by cerebellar ataxia in combination with additional clinical features like optic atrophy, ophthalmoplegia, bulbar and extrapyramidal signs, peripheral neuropathy and dementia. SCA2 is caused by expansion of a CAG repeat in the coding region of the ATXN2 gene. This locus has been mapped to chromosome 12, and it has been determined that the diseased allele contains 37-50 CAG repeats, compared to 17-29 in the normal allele. Longer expansions result in earlier onset of the disease. Alternatively spliced transcript variants encoding different isoforms have been identified for ATXN2.

### **Other Names:**

Ataxin-2, Spinocerebellar ataxia type 2 protein, Trinucleotide repeat-containing gene 13 protein, ATX2, SCA2, TNRC13

### **Source and Purity:**

Rabbit polyclonal antibodies were produced by immunizing animals with a GST-fusion protein containing the middle region of human ATXN2. Antibodies were purified by affinity purification using immunogen.

### **Storage Buffer and Condition:**

Supplied in 1 x PBS (pH 7.4), 100 ug/ml BSA, 40% Glycerol, 0.01% NaN<sub>3</sub>. Store at -20 °C. Stable for 6 months from date of receipt.

### **Species Specificity:**

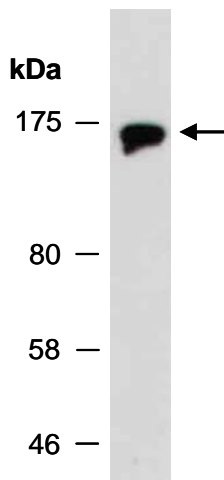
Human, Mouse

### **Tested Applications:**

WB: 1:1,000-1:3,000 (detect endogenous protein\*)

\*: The apparent protein size on WB may be different from the calculated M.W. due to modifications.

**Product Data:**



**Fig 1.** Western blot of total cell extracts from mouse brain, using anti-ATXN2 (M) (R1561-2) at RT for 2 h.