

## Rabbit Anti-Glutathione Synthetase/AF350 Conjugated antibody

SL11850R-AF350

<b>Product Name</b>	Anti-Glutathione Synthetase/AF350
<b>Chinese Name</b>	AF350 标记的谷胱甘肽合成酶抗体
<b>Alias</b>	Glutathione synthase; GSH S; GSH synthetase; GSH-S; GSHB_HUMAN; GSHS; GSS; MGC14098; OTTHUMP00000030711.
<b>Research Area</b>	Tumour Cell biology Neurobiology Signal transduction The new supersedes the old
<b>Immunogen Species</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>React Species</b>	Human,Mouse,Rat(predicted:Dog,Pig,Cow,Horse,Sheep) IF=1:100-500
<b>Applications</b>	not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
<b>Molecular weight</b>	52kDa
<b>Form</b>	Lyophilized or Liquid
<b>Concentration</b>	1mg/ml
<b>immunogen</b>	KLH conjugated synthetic peptide derived from human Glutathione Synthetase
<b>Lsotype</b>	IgG
<b>Purification</b>	affinity purified by Protein A
<b>Storage Buffer</b>	1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol
<b>Storage</b>	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
<b>Product Detail</b>	<b>background:</b> GSS (Glutathione synthetase) is a 474 amino acid protein encoded by the gene located at human chromosome 20q11.2. GSS consists of three loops projecting from an antiparallel $\beta$ -sheet, a parallel $\beta$ -sheet and a lid of anti-parallel sheets,

which provide access to the ATP-binding site. Although Southern blot and gene analysis suggest that GSS may be the only member of a unique family, the crystal structure indicates that GSS belongs to the ATP-GRASP superfamily. GSS is expressed in hemocytes and nucleated cells, including the brain. GSS occurs as a homodimer. There are two steps in the production of Glutathione, beginning with GSS (Glutathione synthetase) is a 474 amino acid protein encoded by the gene located at human chromosome 20q11.2. GSS consists of three loops projecting from an antiparallel  $\beta$ -sheet, a parallel  $\beta$ -sheet and a lid of anti-parallel sheets, which provide access to the ATP-binding site. Although Southern blot and gene analysis suggest that GSS may be the only member of a unique family, the crystal structure indicates that GSS belongs to the ATP-GRASP superfamily. GSS is expressed in hemocytes and nucleated cells, including the brain. GSS occurs as a homodimer. There are two steps in the production of Glutathione, beginning with  $\gamma$ -GCS and ending with GSS. In an ATP-dependent reaction, GSS produces Glutathione from  $\gamma$ -glutamylcysteine and glycine precursors. Partial hepatectomy, diethyl maleate, buthionine sulfoximine, tert-butylhydroquinone and thioacetamide increase the expression of GSS, which causes an increase in Glutathione levels. An inherited autosomal recessive disorder, 5-oxoprolinuria (pyroglutamic aciduria), is caused by GSS deficiencies, which leads to central nervous system damage, hemolytic anemia, metabolic acidosis and urinary excretion of 5-oxoproline. A missense mutation in the gene encoding GSS leads to a GSS deficiency restricted to erythrocytes, which causes only hemolytic anemia.  $\gamma$ -GCS and ending with GSS. In an ATP-dependent reaction, GSS produces Glutathione from  $\gamma$ -glutamylcysteine and glycine precursors. Partial hepatectomy, diethyl maleate, buthionine sulfoximine, tert-butylhydroquinone and thioacetamide increase the expression of GSS, which causes an increase in Glutathione levels. An inherited autosomal recessive disorder, 5-oxoprolinuria (pyroglutamic aciduria), is caused by GSS deficiencies, which leads to central nervous system damage, hemolytic anemia, metabolic acidosis and urinary excretion of 5-oxoproline. A missense mutation in the gene encoding GSS leads to a GSS deficiency restricted to erythrocytes, which causes only hemolytic anemia.

**Function:**

Sulfur metabolism; glutathione biosynthesis; glutathione from L-cysteine and L-glutamate: step 2/2.

**Subunit:**

Homodimer.

**DISEASE:**

Defects in GSS are the cause of glutathione synthetase deficiency (GSS deficiency) [MIM:266130]; also known as 5-oxoprolinuria or pyroglutamic

aciduria. It is a severe form characterized by an increased rate of hemolysis and defective function of the central nervous system.

Defects in GSS are the cause of glutathione synthetase deficiency of erythrocytes (GLUSYNDE)[MIM:231900]. Glutathione synthetase deficiency of erythrocytes is a mild form causing hemolytic anemia.

**Similarity:**

Belongs to the eukaryotic GSH synthase family.

**Database links:**

[Entrez Gene: 2937](#) Human

[Entrez Gene: 14854](#) Mouse

[Entrez Gene: 25458](#) Rat

[Omim: 601002](#) Human

[SwissProt: P48637](#) Human

[SwissProt: P51855](#) Mouse

[SwissProt: P46413](#) Rat

[Unigene: 82327](#) Human

[Unigene: 252316](#) Mouse

[Unigene: 1692](#) Rat

**Important Note:**

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.