

Product Information

Anti-Folic acid antibody, Mouse monoclonal

Clone VP-52, purified from hybridoma cell culture

Product Number **SAB4200793**

Product Description

Monoclonal Anti-Folic acid (mouse IgG2b isotype) is derived from the VP-52 hybridoma, produced by the fusion of mouse myeloma cells and splenocytes from a mouse immunized with 5-methyltetrahydrofolic acid (5MTHFA), conjugated to KLH. The isotype is determined by ELISA using Mouse Monoclonal Antibody Isotyping Reagents, Product Number ISO2. The antibody is purified from the culture supernatant of the hybridoma cells.

Monoclonal Anti-Folic acid recognizes folate and an epitope present on both the biologically active analog 5MTHFA and folic acid. The antibody reacts with folic acid or 5MTHFA either free or bound to a carrier, such as KLH or BSA. The antibody binds folate in human plasma and serum (when it is bound to the endogenous folate binder). The antibody does not cross react with tetrahydrofolic (THFA), folinic acid (FNA), dihydrofolic acid (FAH₂, citrovorum factor, leucovorin). The antibody is recommended for use in various immunological techniques, including ELISA and immunoblotting.¹

Folic acid (pteroylglutamic acid) and Vitamin B₁₂ (cobalamin) are essential constituents for normal growth of mammalian cells. The normal serum range is 3-16 ng/mL for folate and 0.2-0.9 ng/mL for vitamin B₁₂. Folic acid plays an important role in cellular metabolic activities such as functioning as a cofactor in the one-carbon metabolism for DNA and RNA synthesis as well as nucleotide and amino acid biosynthesis. A lack of folate or folic acid nutrition can lead to folic acid deficiency and result in several health problems, including macrocytic anemia, elevated plasma homocysteine, cardiovascular disease, birth defects, carcinogenesis, muscle weakness, and walking difficulty.² These deficiencies may appear in pregnant women, alcoholics, those whose diets do not include raw fruits and vegetables, and people with structural or functional damage to the small intestine.

Vitamin B₁₂ and folic acid are metabolically interrelated. In the absence of vitamin B₁₂, 5MTHFA cannot be converted to tetrahydrofolic acid and enter the metabolic pool of 1-carbon fragment acceptors. Since this is the only known metabolic pathway involving 5MTHFA in humans, B₁₂ depletion will cause a decrease in the availability of other folic acid derivatives required for miscellaneous biosynthetic pathways, including thymidylate synthetase, an enzyme necessary for DNA synthesis.

Vitamin B₁₂ and folate deficiencies are the most common causes of megaloblastic anemia, abnormal hemopoiesis, interference in the maintenance of normal nerve tissue, and general intracellular uptake and function disorders in humans.³ It is hematologically and clinically indistinguishable thus it is necessary to determine the level of vitamin B₁₂ in the serum and folate in both the serum and red blood cells to establish the etiology of the megaloblastic anemia and treatment respectively.

Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide as a preservative.

Antibody Concentration: ~1.0 mg/mL

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2–8 °C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

Indirect ELISA: a working concentration of 0.25-0.5 µg/mL is recommended using 3 µg/mL folic acid conjugated to a carrier for coating.

Note: In order to obtain best results in different techniques and preparations, it is recommended to determine optimal working concentration by titration test.

References

1. Destito, G. et al., *Chem. Biol.*, **14**, 1152-62 (2007).
2. Hwang, S.Y. et al., *J. Cell Physiol.*, **233**, 736-47 (2018).
3. Halsted, C.H., *Annu. Rev. Med.*, **31**, 79-87 (1980).

SG,DR,OKF,LV,MAM 04/18-1