

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse FGF-23 in direct ELISAs and Western blots. In direct ELISAs, this antibody shows approximately 50% cross-reactivity with recombinant human (rh) FGF-23. In Western blots, this antibody does not cross-react with rhFGF-3, -4, -5, -7, -9, -10, -11, -12, -13, -16, -17, -18, -19, acidic, basic, rmFGF-8b, -8c, -15, or -21.
Source	Monoclonal Rat IgG _{2A} Clone # 283507
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse FGF-23 Tyr25-Val251 (Arg179Gln) Accession # Q9EPC2
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

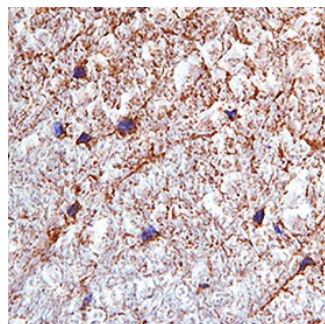
APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Immunohistochemistry	8-25 µg/mL	See Below

DATA

Immunohistochemistry



FGF-23 in Mouse Brain. FGF-23 was detected in perfusion fixed frozen sections of mouse brain (cortex) using Rat Anti-Mouse FGF-23 Monoclonal Antibody (Catalog # MAB26291) at 15 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Rat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS017) and counterstained with hematoxylin (blue). Specific staining was localized to glial cells. View our protocol for [Chromogenic IHC Staining of Paraffin-embedded Tissue Sections](#).

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

Fibroblast growth factor 23 (FGF-23) is a 30-32 kDa member of the FGF gene family. Based on its structure, it is further classified as an FGF19 subfamily member. This subfamily includes FGF-19, -21, and -23. Like all other FGF subfamilies, FGF-19 subfamily members contain a 120 amino acid (aa) core FGF domain that exhibits a β -trefoil structure (1, 2). Unlike other FGF subfamilies, FGF-19 subfamily members exist as highly diffusible molecules that is attributed to poor ECM/heparin sulfate binding (3-6). The cDNA for mouse FGF-23 predicts a 251 aa polypeptide that contains a 24 aa signal sequence and a 227 aa mature region (7). Mature mouse FGF-23 shows 72% aa identity to human FGF-23 (8). The FGF-19 subfamily shares an unusual receptor configuration. The standard model for FGF signaling requires an FGF:FGF R:heparin sulfate complex. Given FGF-23's minimal association with heparin, a substitute termed (α -) Klotho has evolved that serves the same function. Although FGF-23 binds to the widely expressed "c" isoforms of FGF R1 and 3 plus FGF R4, Klotho has a restricted distribution that limits FGF-23 activity (10-12). It should be noted that heparin-dependency has been reported for FGF-19 signaling, and this observation may extend to FGF-23 (13). The FGF-19 subfamily is considered endocrine in nature. All three subfamily members impact some aspect of metabolism and all three are induced by a nuclear receptor heterodimer that includes the retinoid X receptor (14-16). FGF-23 is considered a phosphatonin; that is, a molecule that reduces circulating plasma phosphate. It is produced by osteocytes and osteoblasts in response to high circulating phosphate levels, elevated parathyroid hormone that induces hypercalcemia, and circulatory volume loading. Upon binding to FGF-23 receptors on renal proximal tubular epithelium, two basic changes are seen. First, the enzyme responsible for generating the active form of vitamin D is suppressed, resulting in decreased levels of bioactive vitamin D. Since vitamin D promotes intestinal phosphate absorption, plasma phosphate declines. Second, the transporters responsible for phosphate resorption on renal epithelium are down regulated, resulting in decreased uptake from urine and again a decline in blood phosphorus (17, 18).

References:

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