The natriuretic peptides are a group of structurally similar peptides that are genetically distinct and play a role in several processes, including cardiovascular, renal and endocrine homeostasis. The atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are derived from myocardial cell origin and are cardiomyocytes secreted from the atrium and ventricle of the heart, respectively. The C-type natriuretic peptide (CNP) is derived from endothelial cell origin and acts as an endothelium-derived relaxing factor (EDRF). These peptides mediate their effects through three receptors. NPR-A (also designated GC-A) binds both ANP and BNP, which stimulates 3', 5'-cyclic guanosine monophosphate (cGMP) to mediate natriuresis, vasodilation, renin inhibition, antigrowth and lusitropic properties. NPR-B (also designated GC-B) binds CNP and also stimulates cGMP to facilitate vasodilation and growth inhibition. NPR-C, also designated the “clearance” receptor, clears all three peptides, which are subsequently degraded by the ectoenzyme neutral endopeptidase. The natriuretic peptide system plays an important role in hypertension, congestive heart failure, as well as in the regulation of renal and endocrine homeostasis. The atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are derived from myocardial cell origin and are cardiomyocytes secreted from the atrium and ventricle of the heart, respectively. The C-type natriuretic peptide (CNP) is derived from endothelial cell origin and acts as an endothelium-derived relaxing factor (EDRF). These peptides mediate their effects through three receptors. NPR-A (also designated GC-A) binds both ANP and BNP, which stimulates 3', 5'-cyclic guanosine monophosphate (cGMP) to mediate natriuresis, vasodilation, renin inhibition, antigrowth and lusitropic properties. NPR-B (also designated GC-B) binds CNP and also stimulates cGMP to facilitate vasodilation and growth inhibition. NPR-C, also designated the “clearance” receptor, clears all three peptides, which are subsequently degraded by the ectoenzyme neutral endopeptidase. The natriuretic peptide system plays an important role in hypertension, congestive heart failure, atherosclerosis and renal diseases, and may be a therapeutic target in the treatment of these diseases.

REFERENCES

CHROMOSOMAL LOCATION
Genetic locus: NPR3 (human) mapping to 5p13.3; Npr3 (mouse) mapping to 15 A1.

SOURCE
NPR-C (E-5) is a mouse monoclonal antibody raised against amino acids 141-440 of NPR-C of human origin.

PRODUCT
Each vial contains 200 µg IgG1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS
NPR-C (E-5) is recommended for detection of NPR-C of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation (1-2 µg per 100-500 µg of total protein [1 ml of cell lysate]), immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).


Molecular Weight of NPR-C: 64-66 kDa.

Positive Controls: NPR-C (m): 293T Lysate: sc-122112 or HeLa whole cell lysate: sc-2200.

RECOMMENDED SUPPORT REAGENTS
To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgG HRP: sc-516102 with DAB, 50X: sc-24982 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use m-IgG HRP FITC: sc-516140 or m-IgG HRP PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850. 4) Immunohistochemistry: use m-IgG HRP: sc-516102 with DAB, 50X: sc-24982 and Immunohistomount: sc-45086, or Organo/Limonene Mount: sc-45087.

DATA

STORAGE
Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE
For research use only, not for use in diagnostic procedures.