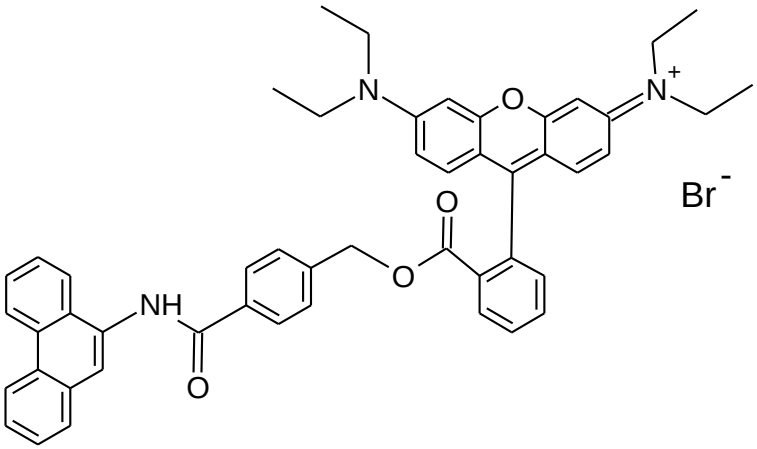
PRODUCT INFORMATION

RPAC

Cat. No. ME046.1 (1 mg)
Cat. No. ME046.2 (5 mg)

Rhodamine B-[(phenanthren-9-yl)-aminocarbonyl]benzyl ester



- Iron-insensitive control for the mitochondrial fluorescent iron indicator RPA
- Mitochondria specific
- Non-toxic

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Product Information

RPAC is used as a iron-insensitive control for the structurally similar mitochondrial fluorescent iron indicator RPA [1, 3]. RPAC [2], which consists of the same fluorophore and linker as RPA has no iron-chelating capacity but shows the same selective mitochondrial accumulation. Parallel incubation of cells loaded with RPA or RPAC showed similar fluorescence recordings.

Product Data

product name:	RPAC
function:	fluorescent, non iron chelating, “negative” control
chemical name:	Rhodamine B-[(phenanthren-9-yl)amino-carbonyl]benzyl ester
molecular formula:	$C_{50}H_{46}N_3O_4Br$
molecular weight:	832 g/mol
absorption maximum:	λmax (log ε) = 567 nm
emission maximum:	602 nm
stability:	4°C, stored dry and protected from light
appearance:	purple solid
purity:	> 98% (1H NMR, 500 MHz)
in vitro toxicity:	non toxic

Considerations for Use

RPAC is used in the same manner as RPA[1]. 1-5 mM stock solutions of RPAC in DMSO can be prepared and aliquots should be kept at –20°C. When stored properly at –20°C, the frozen aliquots are stable and can be used for at least 2 – 3 months.

References

1. Selective determination of mitochondrial chelatable iron in viable cells with a new fluorescent sensor. F. Petrat et al. *Biochem. J.* (2002) 362, 137-147
2. Cold-induced apoptosis of hepatocytes: mitochondrial permeability transition triggered by nonmitochondrial chelatable iron. U. Rauen et al. Free Radical Biology & Medicine, Vol. 35, No. 12, pp. 1664-1678, 2003
3. The chelatable iron pool in living cells: A methodically defined quantity. F. Petrat et al. Biol. Chem., Vol. 383, pp. 489-502, 2002
4. Assessment of chelatable mitochondrial iron by using mitochondrion-selective fluorescent iron indicators with different iron-binding affinities. U. Rauen et al. ChemBioChem 2007, 8, 341-352
5. Oxidative inactivation of mitochondrial Aconitase results in iron and H_2O_2-mediated neurotoxicity in rat primary mesencephalic cultures. David Cantu *et al*. PlosOne, September 2009, Vol 4, Issue 9, p 1-9

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