Patent Search

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Abstract:

A LYSOSOME SPECIFIC NITRIC OXIDE PROBE FOR NEUROINFLAMMATION AND PROCESS OF SYNTHESIS THEREOF ABSTRACT The present invention relates to a fluores having a general formula 1which specifically target lysosomes and monitor Nitric oxide (NO) levels in normal and inflamed conditions. The probe potentially identifie activated human microglia cells (HMC3) by turning on its fluorescence signal and concurrently monitored NO during iNOS- induced phagocytosis. Additionally it also e detect SARs-Co-V2 RNA transfected activated human microglia by imaging endogenous NO in lysosomes. Moreover, the present invention also discloses the process f synthesis of the NO probe. Scheme 1

Complete Specification

Description:A LYSOSOME SPECIFIC NITRIC OXIDE PROBE FOR NEUROINFLAMMATION AND PROCESS OF SYNTHESIS THEREOF

FIELD OF INVENTION

The present invention relates to a phagocytosis probe that specifically target lysosomes and monitor NO level in normal and inflamed conditions. The probe detects activated human microglia (HMC3) cells through turn-on fluorescence response and remain silent in non-activated macrophages. Moreover, the present invention a relates to the process for the synthesis of the lysosome specific phagocytosis probe which is a valuable probe for sensing lysosomal NO in immune cells, and for va the vaccine's efficacy for targeting SARS-CoV-2 RNA.

BACKGROUND OF INVENTION

After being recognized as a 'molecule of the year' in 1992, NO has been recognized as a most versatile player in innate immunity and inflammation. It exhibits a ran physiological functions such as regulation of the blood pressure in the endothelial vessel, inhibition of platelet aggregation, repairing of neurotransmission, acute a chronic inflammation, and mainly the host immune defense mechanism. Other than these roles, NO plays essential role in fine tuning of brain microenvironment fo synaptic transmission and brain plasticity, especially in the cortex and hippocampus, and the cellular nitrergic/oxidative stress imposed by dysregulated NO levels h been linked to impaired synaptic plasticity and early onset of neurodegeneration. Considering the role of NO and its production in the immune defense system, the widespread expression of iNOS has been well characterized a long time back. It is implicated that utilizing electrons donated from NADPH, L-arginine, and molecula oxygen synthesized NO by an enzymatic process in iNOS in both the macrophages and endothelial cells to maintain cellular physiological functions. In the immune function, the activated macrophages release a high level of NO initiated by cytokines. However, the overexpression of the NO causes auto-immune disorder by inhib

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