

Datasheet for GP-T065

Guinea Pig Spinal Cord

Overview

Description:	Guinea Pig Spinal Cord - GP-T065
Item No.:	GP-T065
Size:	1 Each
Applications:	Biochemical Assay, Functional Assay, IHC, Other
Origin:	Guinea Pig

Product Details

Species of Origin:	Guinea Pig
--------------------	------------

Application Details

Suggested Applications:	Biochemical Assay, Functional Assay, IHC, Other (Based on references)
-------------------------	---

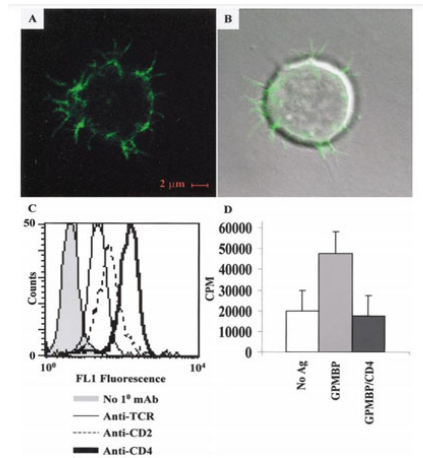
Formulation

Physical State:	Tissue
Sterility:	Non-sterile

Shipping & Handling

Shipping Condition:	Dry Ice
Storage Condition:	Store tissue at -20° C or colder prior to use.
Expiration:	No expiration date is given for this product if properly stored.

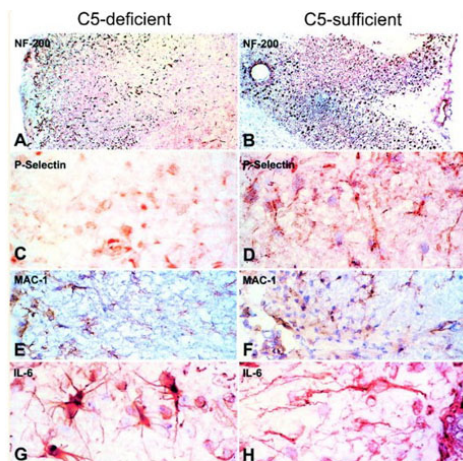
Images



Immunofluorescence Microscopy

Donor R1-trans T-APC have I-A dendrites. R1-trans T-APC were labeled with OX6-FITC (mouse anti-rat I-A mAb). Cells were then examined via confocal microscopy and were viewed in a FITC channel (A) or an overlay of FITC and DIC channels (B). (C) R1-trans T-APC were stained with no primary mAb (shaded line), R73 (anti-TCR) (thin line), OX34 (anti-CD2) (dashed line), or W3/25 (anti-CD4) (boldface line) and a goat anti-mouse FITC-conjugated secondary. (D) R1-trans T-APC were cultured in the presence (gray bar) or in the absence (white bar) of 3.2 M GPMBP with (black bar) or without W3/25 (anti-CD4) for 2 days. GPMBP was purified from guinea pig spinal cords (p/n GP-T065). During the last 24 h of the culture, the cultures were pulsed with 1 µCi of [3 H]thymidine. T cells were harvested onto filters to measure [3 H]thymidine incorporation by scintillation counting.

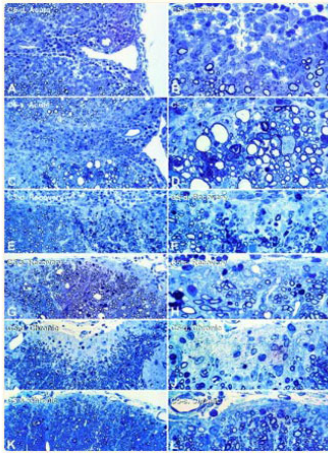
FIG. 2. PMID: 11902826.



Immunohistochemistry

Immunohistochemistry of C5-d and C5-s mice with EAE. EAE was induced by subcutaneous injection of 700 µg purified guinea pig myelin from frozen spinal cords (p/n GP-T065), in incomplete Freund's adjuvant containing 70 µg Mycobacterium tuberculosis and H37RA, followed by intravenous injection with 100 ng Pertussis toxin, on day 0, 2, and 7 post-immunization (p.i.). Neurofilament (NF-200): Sections from C5-d with chronic EAE revealed weaker and scattered staining of NF-200 (A) in areas corresponding to a chronic lesion, compared to the robust, densely-packed staining in sections of C5-s mice (B), where most axons survived. P-Selectin: In chronic EAE, staining for the endothelial adhesion molecule, P-Selectin, was lower in C5-d (C) than C5-s (D), perhaps indicating reduced infiltration of inflammatory cells into the tissue. MAC-1: Increased staining of macrophages in C5-s mice (E), during acute disease suggested enhanced phagocytosis of myelin debris in C5-s, in comparison to C5-d mice (F). IL-6: In acute EAE, IL-6 was enhanced in reactive astrocytes in C5-d (G), in comparison to C5-s mice (H). Magnifications: A and B, ×63; E, F, G, H, ×625.

Fig 4. PMID: 12937147.



Immunohistochemistry

Histopathology of acute, recovery, and chronic phases of EAE in C5-d and C5-s mice. EAE was induced by subcutaneous injection of 700 µg purified guinea pig myelin from frozen spinal cords (p/n GP-T065), in incomplete Freund's adjuvant containing 70 µg Mycobacterium tuberculosis and H37RA, followed by intravenous injection with 100 ng Pertussis toxin, on day 0, 2, and 7 post-immunization (p.i.). Toluidine-blue-stained epoxy sections (1 µm) from the upper lumbar of C5-d mice with acute EAE (A and B), revealed well-demarcated, confined lesions with inflammation and a narrow rim of demyelinated axons. Lesions of C5-s mice with acute EAE (C and D) are more diffuse and display inflammation and demyelination in addition to Wallerian degeneration (D, lower left), dystrophic axons (D, center right), and myelin ovoids (D, left). In the recovery phase of EAE, C5-d mice (E and F, cervical spinal cord) show extensive Wallerian degeneration (dense droplets, lower right) and demyelination. In contrast, extensive remyelination was present in C5-s mice (G and H, S1 spinal cord), shown by numerous thinly myelinated fibers along the meningeal surface (H). Numerous fibrous astrocytes with pale nuclei occur within the remyelinated zone. During chronic EAE, C5-d mice (I and J) developed a prominent zone of intense gliosis at the margin of the spinal cord, from which nerve fibers and oligodendrocytes were depleted. Lesions in C5-s mice (K and L) displayed numerous remyelinated axons at the edge of the spinal cord, where some gliosis is also present.

Fig 3. PMID: 12937147.

References

- Constantinescu CS et al. Astrocytes as antigen-presenting cells: expression of IL-12/IL-23. *J Neurochem.* (2005)
- Mannie MD et al. MHC class II biosynthesis by activated rat CD4+ T cells: development of repression in vitro and modulation by APC-derived signals. *Cell Immunol.* (2004)
- Weerth S et al. Complement C5 in experimental autoimmune encephalomyelitis (EAE) facilitates remyelination and prevents gliosis. *Am J Pathol.* (2003)
- Patel DM et al. Intercellular exchange of class II MHC complexes: ultrastructural localization and functional presentation of adsorbed IA/peptide complexes. *Cell Immunol.* (2001)
- Jewell SD et al. Oral tolerance as therapy for experimental autoimmune encephalomyelitis and multiple sclerosis: demonstration of T cell anergy. *Immunol Cell Biol.* (1998)
- Nagasato, K. et al. Exon 2 containing myelin basic protein (MBP) transcripts are expressed in lesions of experimental allergic encephalomyelitis (EAE). *Journal of Neuroimmunology* (1997)
- Voskuhl RR et al. Gender differences in autoimmune demyelination in the mouse: implications for multiple sclerosis. *Ann Neurol.* (1996)
- Cross AH et al. Long-term inhibition of murine experimental autoimmune encephalomyelitis using CTLA-4-Fc supports a key role for CD28 costimulation. *J Clin Invest.* (1995)
- Kim C et al. Adoptive transfer of murine experimental autoimmune encephalomyelitis in SJL. Thy-1 congenic mouse strains. *J Neuroimmunol.* (1993)
- Ann Neurol. Anti-tumor necrosis factor therapy abrogates autoimmune demyelination. *Selmaj K et al.* (1991)
- Cross AH et al. Adoptive transfer of experimental allergic encephalomyelitis and localization of the encephalitogenic epitope in the SWR mouse. *J Neuroimmunol.* (1991)
- Fuller KA et al. Oral tolerance in experimental autoimmune encephalomyelitis: serum and salivary antibody responses. *J Neuroimmunol.* (1990)
- Bitar DM et al. Suppression of experimental autoimmune encephalomyelitis by the oral administration of myelin basic protein.--- *Cell Immunol.* (1988)
- Badger AM et al. Immunomodulatory activity and non-specific suppressor cell generation by spirogermanium in murine and rat models of cell-mediated immunity. *Immunopharmacology.* (1988)
- Stohl W et al. Detection of the precursor and effector cells of experimental allergic encephalomyelitis in the thoracic duct of the rat. *Cell Immunol.* (1980)

Disclaimer

This product is for research use only and is not intended for therapeutic or diagnostic applications. Please contact a technical service representative for more information. All products of animal origin manufactured by Rockland Immunochemicals are derived from starting materials of North American origin. Collection was performed in United States Department of Agriculture (USDA) inspected facilities and all materials have been inspected and certified to be free of disease and suitable for exportation. All properties listed are typical characteristics and are not specifications. All suggestions and data are offered in good faith but without guarantee as conditions and methods of use of our products are beyond our control. All claims must be made within 30 days following the date of delivery. The prospective user must determine the suitability of our materials before adopting them on a commercial scale. Suggested uses of our products are not recommendations to use our products in violation of any patent or as a license under any patent of Rockland Immunochemicals, Inc. If you require a commercial license to use this material and do not have one, then return this material, unopened to: Rockland Inc., P.O. BOX 5199, Limerick, Pennsylvania, USA.

