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The Role of Death Domains Superfamily in Multiple Sclerosis Pathogenesis

Kabir M. Hamid[1], Abbas Mirshafiey[2*]

[1]Department of Immunology, School of Public Health, Tehran University of Medical Sciences—International Campus (TUMS-IC), Tehran, Iran
[2]Department of Immunology, School of Public Health, Tehran University of Medical Sciences (TUMS), Tehran, Iran
Email: kabiru20@yahoo.com, [*]mirshafiey@tums.ac.ir

Received 2 November 2014; revised 19 December 2014; accepted 7 January 2015

Abstract

Multiple sclerosis (MS) and its animal model, experimental autoimmune encephalomyelitis (EAE), are inflammatory diseases of the central nervous system (CNS), mediated by several immune cells. Oligodendrocytes are responsible for the formation and maintenance of myelin around multiple axons. In MS oligodendrocytes are the targets of inflammatory and immune attacks. Thus, the destruction of a single oligodendrocyte, possibly by apoptosis, results in the loss of myelin around several axons and the loss of many oligodendrocytes limiting the ability to repair or regenerate demyelinated areas. Apoptosis is mediated by an aggregation of various protein components, specifically death domains (DD) superfamily. This superfamily is composed of the death domain (DD), the death effector domain (DED), the caspase recruitment domain (CARD) and the pyrin domain (PYD) subfamilies. Within each subfamily, members form homotypic interactions and facilitate the assembly of oligomeric signaling complexes. Members of the death domain superfamily are critical components of apoptotic and inflammatory signaling. We summarize the structure and functions of the DD superfamily, and describe the role of the DD proteins in oligodendrocytes death and proinflammatory activation in MS pathogenesis.

Keywords

Multiple Sclerosis, Death Domain, Oligodendrocytes, Inflammation, Apoptosis

Subject Areas: **Immunology, Neurology, Pathology**

1. Introduction

Multiple sclerosis (MS) and its animal model, experimental autoimmune encephalomyelitis (EAE), are inflam-

[*]Corresponding author.

How to cite this paper: Hamid, K.M. and Mirshafiey, A. (2015) The Role of Death Domains Superfamily in Multiple Sclerosis Pathogenesis. *Open Access Library Journal*, **2**: e1210. http://dx.doi.org/10.4236/oalib.1101210