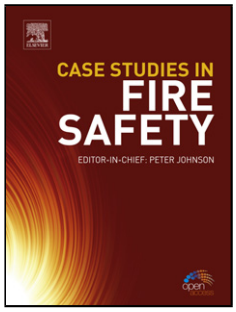

Title: XPS investigations on the corrosion mechanism of V(IV) conversion coatings on hot-dip galvanized steel

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Immune Evasion of the CD1d/NKT Cell Axis

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Highlights:

- Viruses, bacteria and tumors can impair the CD1d/NKT cell axis
- CD1d can be downregulated or cell signaling pathways disrupted
- NKT cells can be directly infected and/or unable to recognize CD1d
- Immune evasion strategies might be used to develop new treatments paradigms

ABSTRACT

Many reviews on the CD1d/NKT cell axis focus on the ability of CD1d-restricted NKT cells to serve as effector cells in a variety of disorders, be they infectious diseases, cancer or autoimmunity. In contrast, here, we discuss the ways that viruses, bacteria and tumor cells can evade the CD1d/NKT cell axis. As a result, these disease states have a better chance to establish a foothold and potentially cause problems for the subsequent adaptive immune response, as the host tries to rid itself of infections or tumors.

Introduction

Classical antigen presentation in the cellular adaptive immune response occurs via the recognition of peptides presented by the major histocompatibility complex (MHC) class I or class II molecules, to conventional T lymphocytes [1,2]. In contrast, the MHC class I-like CD1d molecule presents lipids to natural killer T (NKT) cells [3]. Invariant NKT cells are defined as those CD1d-specific T cells that have an invariant T cell α chain rearrangement (Vα14-Jα18 in mouse; Vα24-Jα18 in humans). As part of the innate immune response, the CD1d/NKT cell axis has been shown to play various protective roles in anti-microbial and anti-tumor host defense [4-8]. However, several pathogens and tumor cells have various means to impair antigen (Ag) presentation by CD1d and/or NKT cell function.

CD1d acquires the lipid antigens it presents by an intracellular mechanism (Figure 1). CD1d molecules are synthesized in the endoplasmic reticulum and are loaded with a non-antigenic lipid [3,9,10]. Like other glycosylated proteins, they then traverse through the Golgi and are ultimately expressed on the cell surface [11]. However, these CD1d molecules are not loaded with a lipid that can stimulate NKT cells. Instead, thanks to a tyrosine-based endosomal targeting sequence [12], CD1d molecules re-enter the cells and traffic through late endocytic compartments, where the non-antigenic lipid is replaced by one that is. Upon re-expression on