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A Review of the Role of Cytokines in the Immune System

Zhendong CK Tay*

Department of Biological Sciences, National University of California, USA

Abstract

It has been demonstrated that interleukin-1 beta (IL-1), which is known to play a role in coordinating the physiological and behavioural changes that take place during illness, has a major impact on memory consolidation. We provide neurobiological evidence to back up this claim, showing that there are substrates for IL-1 to affect memory function and brain plasticity. Then, we provide behavioural proof that central IL-1 administration and substances that stimulate central IL-1 activity hinder the consolidation of memories that depend on the development of the hippocampus formation but have no impact on the consolidation of memories that are hippocampal-independent. Furthermore, we show that blocking the effects of IL-1 prevents the defects in hippocampal-dependent memory consolidation caused by drugs that stimulate IL-1 activity. We examine these findings' implications for a physiological role in the final section.

Keywords: Anti-inflammatory; Pro-inflammatory; Toll-like receptors; Immunofluorescence assays

Introduction

According to their functions, the cytokines have been categorised as pro- or anti-inflammatory. IL-10 and TGF- (transformation growth factor-beta) are the two main anti-inflammatory cytokines, and they may, among other things, limit the production of pro-inflammatory cytokines14 [14]. We can name IL-1, IL-2, IL-12, IL-18, IFN- and TNF- as pro-inflammatory cytokines. Some competitive antagonists, such the IL-1 receptor antagonist (IL-1ra), which blocks IL-1 from binding to its receptor15, are considered to have anti-inflammatory properties. A subunit of IL-12 known as p40, which is known to be a pro-inflammatory cytokine (IL-12)14, can block IL-12 activity when it is released, which indirectly has an anti-inflammatory effect (16). By preventing the synthesis of IL-1217, chemokine, a chemotactic protein of monocytes (MCP-1), can also serve as an indirect anti-inflammatory. Cell surface glycoconjugates are believed to be crucial for a number of biological processes, including cell-cell and cell-substrate interactions, bacterial adhesion, cell immunogenicity, and cell signalling. Glycosylation is one of the most significant changes of proteins and lipids [1].

Glycan structures vary based on the kind of cell, the period of development, and the differentiation of the cell. Pathologic conditions such as malignancies and inflammatory disorders like cystic fibrosis or bowel ailments also modify glycan structures. Cancer-related changes in cell glycosylation mostly impact the outer portion of glycans, resulting in the development of cell surface antigenic structures that are closely linked to poor prognosis in some tumours [2].

Immune system cells are affected by physical exercise

Neutrophils: White blood cells known as neutrophils are very mobile, transient, and heavily populated with secretory granules. They come from the bone marrow, where they develop as a result of the right cytokines. They then leave the bone marrow, enter the circulation, and travel to the tissues. In healthy people, peripheral blood neutrophils account for 40–80% of all white blood cells [13]. The body's largest marginated pool of neutrophils is found in the lungs. Neutrophils play a crucial sentinel role in preserving sterility in the airways. Neutrophils play a dual role in innate immunity as a key effector cell. Infections develop from an overgrowth of bacteria and fungus at areas of injury or trauma if neutrophils are not present (as in congenital neutropenia or the more frequent cyclic neutropenia) [3].

Anti-presenting cells: T lymphocytes can only recognise antigens that are exposed on the surface of presenting cells such dendritic cells, macrophages, and B lymphocytes in conjunction with components from the major histocompatibility complex (MHC). Aerobic activity that is prolonged and intensified compromises the presentation of antigens to T lymphocytes, especially for the Th1 inflammatory response, and lowers the expression of Toll-like receptors (TLRs) in macrophages. This anti-inflammatory action prevents the typical tissue damage brought on by inflammatory mediators and lowers the likelihood of chronic inflammatory disorders, but it also makes people more vulnerable to intracellular microbial infections [4].

NK cell: The NK cells are lymphocytes that naturally kill virus- and tumor-infected cells while ignoring initial sensibilization and being unaffected by MHC presentation. The constant region (Fc) of IgG, the Fcg (CD16), and a neural cell adhesion molecule (CD56), which is in charge of homotypic adhesion52, are all surface identifiers for these cells' receptor III. These cells can be divided into two subpopulations based on the CD56 expression: CD56dim, which have high levels of CD16 and are more cytotoxic and account for 90% of the NK cells found in the peripheral circulation; and CD56bright, which have lower or nonexistent CD16 levels and account for about 10% of the total number of circulating NK cells [5].

Role of cytokine in immune system: Communication between immune cells depends heavily on cytokines. As a result, cytokine profiles that fluctuate with age affect the immune system in many different ways. Inflammatory cytokines, which are predominantly macrophage products (first recognised as acute phase responses to bacterial infection, but also linked to other illnesses frequent in old age), have impacts on multiple systems. A pathophysiological process that causes aging-related illnesses and losses in physical function is chronic

***Corresponding author:** Zhendong CK Tay, Department of Biological Sciences, National University of California, USA, E-mail: zhendong..c.k.tay@gmail.com

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inflammation. Frailty, a physiological decrease that occurs with ageing and the concomitant dysregulation of systems, has been compared to the paradigm of immunological dysregulation involving cytokines [6].

Proinflammatory cytokines like IL-6 and TNF-a are known to increase with ageing. Older age is also linked to several cytokines/chemokines. The idea of frailty, a physiological decrease that occurs with ageing and the concomitant dysregulation of systems, has been compared to the paradigm of immunological dysregulation involving cytokines. It would not be practical to review these cytokines/chemokines in great detail due to the complexity of age-associated changes in them. We concentrate on IL-6 and TNF-a (and their effects on age-related disorders), as well as on alterations in T and NK cells [7].

Materials and Methods

Conditions for cell culture with cells: Fresh buffy coats were used to isolate human PBMCs using density gradient centrifugation and Ficoll-Paque (GE Healthcare, Milwaukee, WI, USA). Recombinant IL-2 was added to T cell media (AIM-V + 5% AB serum) used to cultivate PBMCs. They were then activated using TransAct or soluble OKT3. The recommended cultivation methods were used with human tumour cell lines from ATCC [8].

Analysis using flow cytometry and cytotoxicity assay: Using the Accuri C6 cytometer, flow-cytometric analysis was carried out. A non-radioactive technique was used to assess the cytolytic activity of CAR-modified T cells. Through normal laboratory testing, it is now possible to effectively screen for an IEI [12]. This method is particularly straightforward in analysing the humoral immune system since it measures blood immunoglobulin levels before evaluating the ability to create certain antibodies in response to immunisation. The outcome of both protein and carbohydrate antigen responses should be examined in these later research. Any condition that results in a dysfunctional organ should be clearly identified by this in vivo functional evaluation [9].

Result

At 24 hours, only TLR-2 was expressed by C.albicans, but A.flavus had considerable expression of TLR-1,-2,-6,-7, and -9. TLR-1 and TLR-9 expression, however, were significantly expressed by C. albicans at 3 hours. A. flavus was reported to generate proinflammatory cytokines, mainly interleukin-8 (IL-8), IL-6, and TNF- at 12 hours, while C. albicans expressed IL-6 at 3 hours. A. flavus was found to have much higher levels of IL-1 and IL-17 after 24 hours, however both A. flavus and C. albicans also expressed IL-10 at this time, in addition to MMP-2 and MMP-9. ELISA and/or Immunofluorescence assays were used to confirm that certain immune mediators were expressed [10-12].

Conclusion

The regulation and repair of brain growth depend on neurotropic cytokines and their signalling pathways. As a result, they might end up becoming the focus of future therapeutic interventions during neurodegenerative processes brought on by illnesses, toxins, or trauma. Many of the physiological and behavioural changes that take place during illness are caused by IL-1, which has been well-documented in this regard. According to recent data, elevated levels of IL-1 have a negative impact on memory consolidation processes. The current data point towards this conclusion because even after a conditioning session has ended, environmental triggers or events that produce IL-1 activity impair contextual fear conditioning.

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