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13632. [Chemokines in TBI Chemokines \(CCL\) are chemotactic inflammatory proteins that mediate interactions among inflammatory cells and target cells. In general, chemokines are typically 10 KDa or smaller. CCLs are synthesized and/or released along with other mediator molecules by a variety of cell types that include: astrocytes, microglia, macrophages, eosinophils, neutrophils, dendritic cells, mast cells and natural killer cells \(NK cells\) \[93–95\]. Release of chemokines serves to chemotactically guide receptor-sensitive cells, primarily through activation of G protein-coupled receptors \[96\]. Similar to cytokines, chemokines can be either pro- and/or anti-inflammatory. After a TBI, chemokines contribute to the attraction of a wide range of immune cells to the site of damage \[97,98\]. The specific activities of classes of chemokines have been elucidated following TBIs \[99–101\]. Although a full discussion is beyond the scope of this review, one example, CXC chemokines, activates the migration of neutrophils to the site of the lesion \[96\]. Alternatively, chemokines CCL2, CCL3, CCL5, CCL7, CCL8, CCL13, CCL17 and CCL22 attract monocytes and macrophages \[102–104\]. Other chemokines, such as, CCL1, CCL2, CCL17 and CCL22, are involved in the recruitment of T-lymphocytes \[103\]. Another important role of cytokine and chemokine release is to activate pattern recognition receptors \(PRRs\). PRRs are proteins of the innate immune system and identify danger-associated molecular patterns \(DAMPs\) of cellular stress. This identification, and the ensuing response, helps defend against cell and/or tissue damage \[105,106\]. The PRRs are divided into](#)

several subgroups, depending on cell localization, type and function. One such group is the nucleotide-binding domain leucine-rich repeats (NLRs) [107], which are also called the nucleotide oligomerization domain (NOD)-like receptors. These receptors are located in the cytoplasm and help to regulate the host inflammatory, apoptotic and innate immune responses [108,109]. The NLR family of proteins can be activated by multiple types of cell/tissue damage that are seen in TBI and can form multi-protein complexes called “inflammasomes” [110]. The unique compositions of these inflammasomes depend on the extent and type of cell and tissue damage. Some reports suggested the specific contributions of NLR family proteins (NLRP3-inflammasome) after TBI [110,111]. The NLRP3-inflammasome has been detected in neurons, astrocytes and microglia in the cortex after TBI [110], and the NLRP3-inflammasome complex is associated with increases in the aforementioned IL1- $\beta$  and IL18 [108,110,112]. Interestingly, the NLRP3-inflammasome has also been demonstrated to associate with other CNS inflammatory disorders, including Alzheimer’s disease (AD) [113], which is an increased risk factor following TBI. Thus, the overall contributions of the cytokines and chemokines released after TBI are mediated by the release, and subsequent recruitment of immune cells to the site of injury, and to coordinate the ensuing activity of these cells. These immune cells are an essential part of the innate immune response and also the putative transition to the adaptive immune response and will be discussed below. - Google Search

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patients responded, some did not Less than 3.5 : Most patients did not respond  
The researchers concluded that Acclidine is best suited for patients with a ratio  
of IGF-BP-3/IGF-1 greater than 3.5. Patients with a ratio greater than 3.5 have  
a IGF-1 deficiency. In the case the ratio is less than 3.5, but the concentration  
of IGF-1 is higher than normal, it might be useful to begin with a low dose of  
Acclidine, 1-2 capsules per day. Methods: 90 individuals suffering from CFS  
(Holmes and/or Fukuda criteria) entered the study. They were not allowed to  
take any other medication than minor pain relievers and homeopathic  
medication. The treatment protocol consisted of first 4 weeks: 250 mg four  
times a day in combination with amino acids; second 4 weeks 250 mg  
Acclidine twice daily in combination with amino acids. The primary efficacy  
variable was the change from baseline in CFS as measured by the Clinician's  
Global Impression Scale (CGI) after 4 and 8 weeks of treatment. Secondary  
endpoints were: IGF-1, IGF-BP3 values, LMW/HMW RNase L ratio, GHQ-30,  
POMS, SF-36 and SCL-90. Conclusions: Acclidine in combination with amino  
acids gives an improvement in CFS symptoms (54% in the active group  
compared to 16% in the placebo group). No major adverse events were noted.  
Acclidine appears to be a safe and adequate drug for CFS. This study was  
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