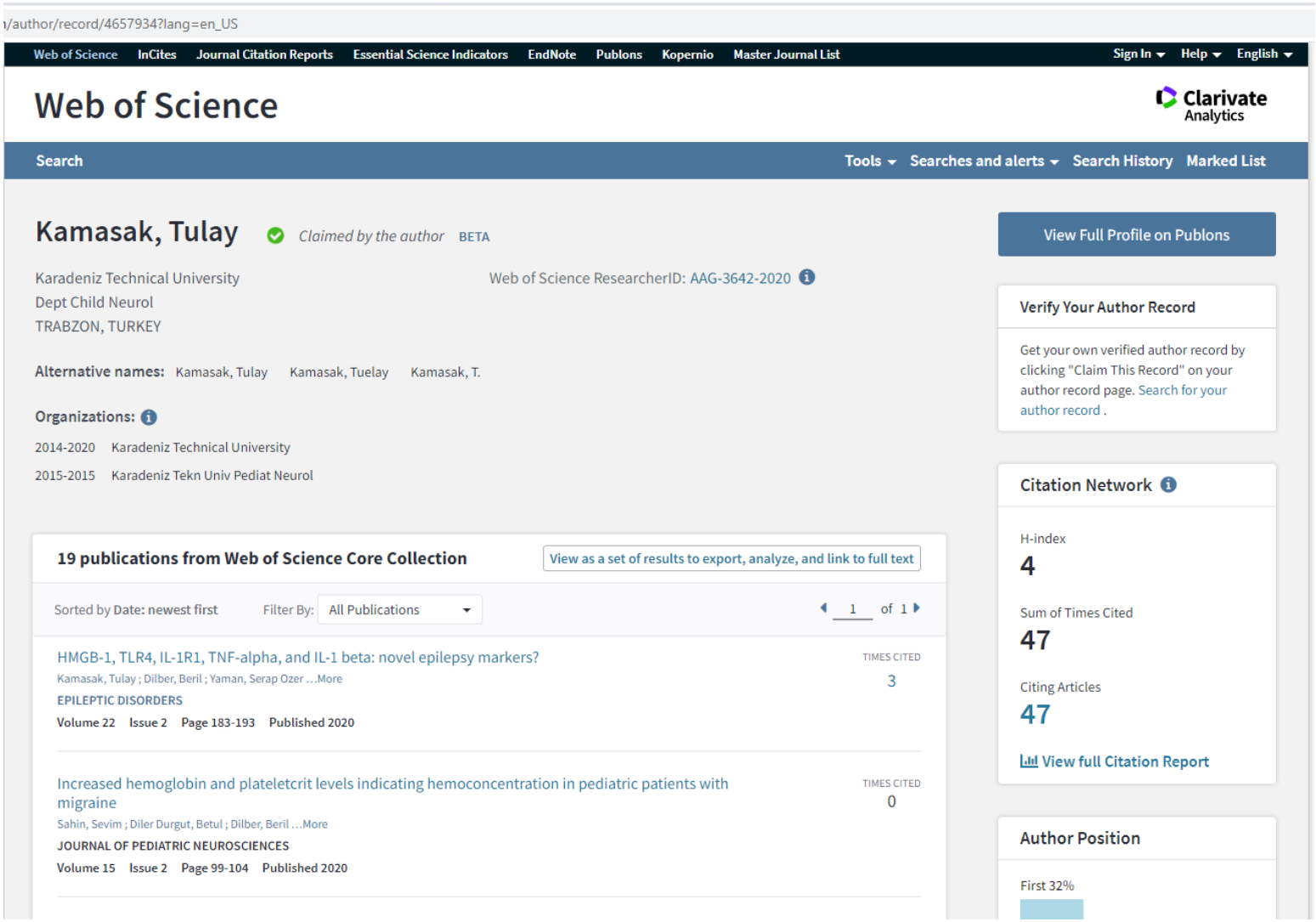
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1. **Emerging Role of Microglia-Mediated Neuroinflammation in Epilepsy after Subarachnoid Hemorrhage**

By: Wang, Jun; Liang, Jingxue; Deng, Jiahong; et al.

MOLECULAR NEUROBIOLOGY

Early Access: JAN 2021

Close Abstract

Epilepsy is a common and serious complication of subarachnoid hemorrhage (SAH), giving rise to increased morbidity and mortality. It's difficult to identify patients at high risk of epilepsy and the application of anti-epileptic drugs (AEDs) following SAH is a controversial topic. Therefore, it's pressingly needed to gain a better understanding of the risk factors, underlying mechanisms and the optimization of therapeutic strategies for epilepsy after SAH. Neuroinflammation, characterized by microglial activation and the release of inflammatory cytokines, has drawn growing attention due to its influence on patients with epilepsy after SAH. In this review, we discuss the risk factors for epilepsy after SAH and emphasize the critical role of microglia. Then we discuss how various molecules arising from pathophysiological changes after SAH activate specific receptors such as TLR4, NLRP3, RAGE, P2X7R and initiate the downstream inflammatory pathways. Additionally, we focus on the significant responses implicated in epilepsy including neuronal excitotoxicity, the disruption of blood-brain barrier (BBB) and the change of immune responses. As the application of AEDs for seizure prophylaxis after SAH remains controversial, the regulation of neuroinflammation targeting the key pathological molecules could be a promising therapeutic method. While neuroinflammation appears to contribute to epilepsy after SAH, more comprehensive experiments on their relationships are needed.

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2. **The performance of the alarmin HMGB1 in pediatric diseases: From lab to clinic**

By: Li, Bo; Peng, Xin; Li, He; et al.

IMMUNITY INFLAMMATION AND DISEASE Volume: 9 Issue: 1 Pages: 8-30 Published: MAR 2021

Early Access: NOV 2020

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Introduction: High-mobility group box protein 1 (HMGB1) is a ubiquitously expressed nonhistone nuclear protein that has multiple functions related to posttranslational modifications and cellular localization. In the nucleus, HMGB1 binds to DNA,

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regulating transcription and determining chromosomal architecture. When released into the extracellular space, HMGB1 activates innate immunity and mediates a wide range of physiological and pathological responses through the differential engagement of multiple surface receptors (i.e., TLR4/2 and RAGE). The effect of HMGB1-mediated inflammatory activation on different systems has received increasing attention. HMGB1 is implicated as a late mediator of sepsis and is also involved in inflammatory and autoimmune diseases. In addition, HMGB1 also affects the occurrence and progression of tumors. However, most studies involving HMGB1 have been focused on adults or mature animals. Due to differences in disease characteristics between children and adults, it is necessary to clarify the role of HMGB1 in pediatric diseases.

Methods and Results: Through systematic database retrieval, this review aimed to first elaborate the characteristics of HMGB1 under physiological and pathological conditions and then discuss the clinical and scientific significance of HMGB1 in the pediatric field according to different systems.

Conclusions: HMGB1 plays an important role in a variety of pediatric diseases and may be used as a diagnostic biomarker and therapeutic target for new strategies for the prevention and treatment of pediatric diseases.

3. **Role of Innate Immune Receptor TLR4 and its endogenous ligands in epileptogenesis**

By: Paudel, Yam Nath; Angelopoulou, Efthalia; Akyuz, Enes; et al.

PHARMACOLOGICAL RESEARCH **Volume: 160** **Article Number: 105172** **Published: OCT 2020**

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Understanding the interplay between the innate immune system, neuroinflammation, and epilepsy might offer a novel perspective in the quest of exploring new treatment strategies. Due to the complex pathology underlying epileptogenesis, no disease-modifying treatment is currently available that might prevent epilepsy after a plausible epileptogenic insult despite the advances in pre-clinical and clinical research. Neuroinflammation underlies the etiopathogenesis of epilepsy and convulsive disorders with Toll-like receptor (TLR) signal transduction being highly involved. Among TLR family members, TLR4 is an innate immune system receptor and lipopolysaccharide (LPS) sensor that has been reported to contribute to epileptogenesis by regulating neuronal excitability. Herein, we discuss available evidence on the role of TLR4 and its endogenous ligands, the high mobility group box 1 (HMGB1) protein, the heat shock proteins (HSPs) and the myeloid related protein 8 (MRP8), in epileptogenesis and post-traumatic epilepsy (PTE). Moreover, we provide an account of the promising findings of TLR4 modulation/inhibition in experimental animal models with therapeutic impact on seizures.

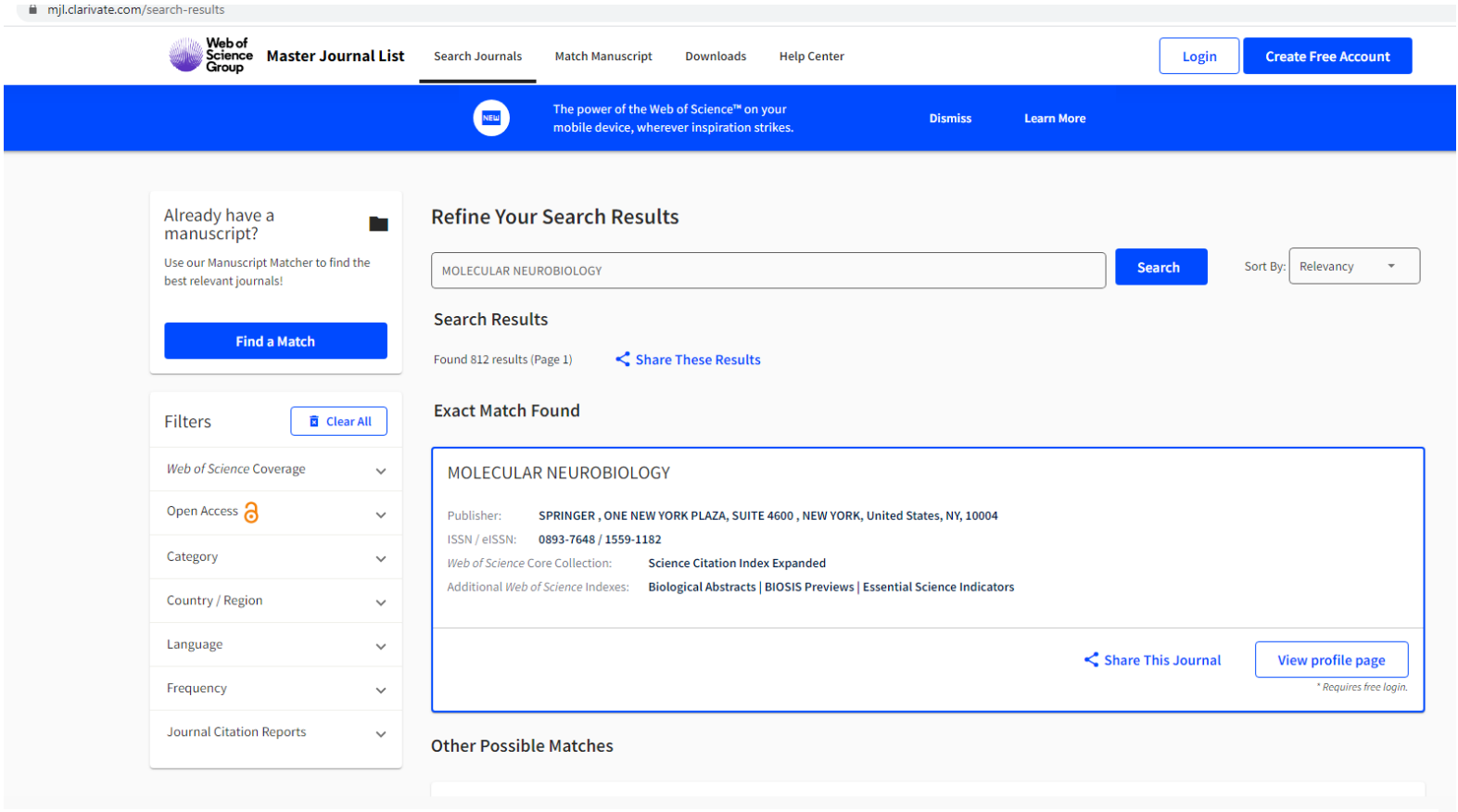
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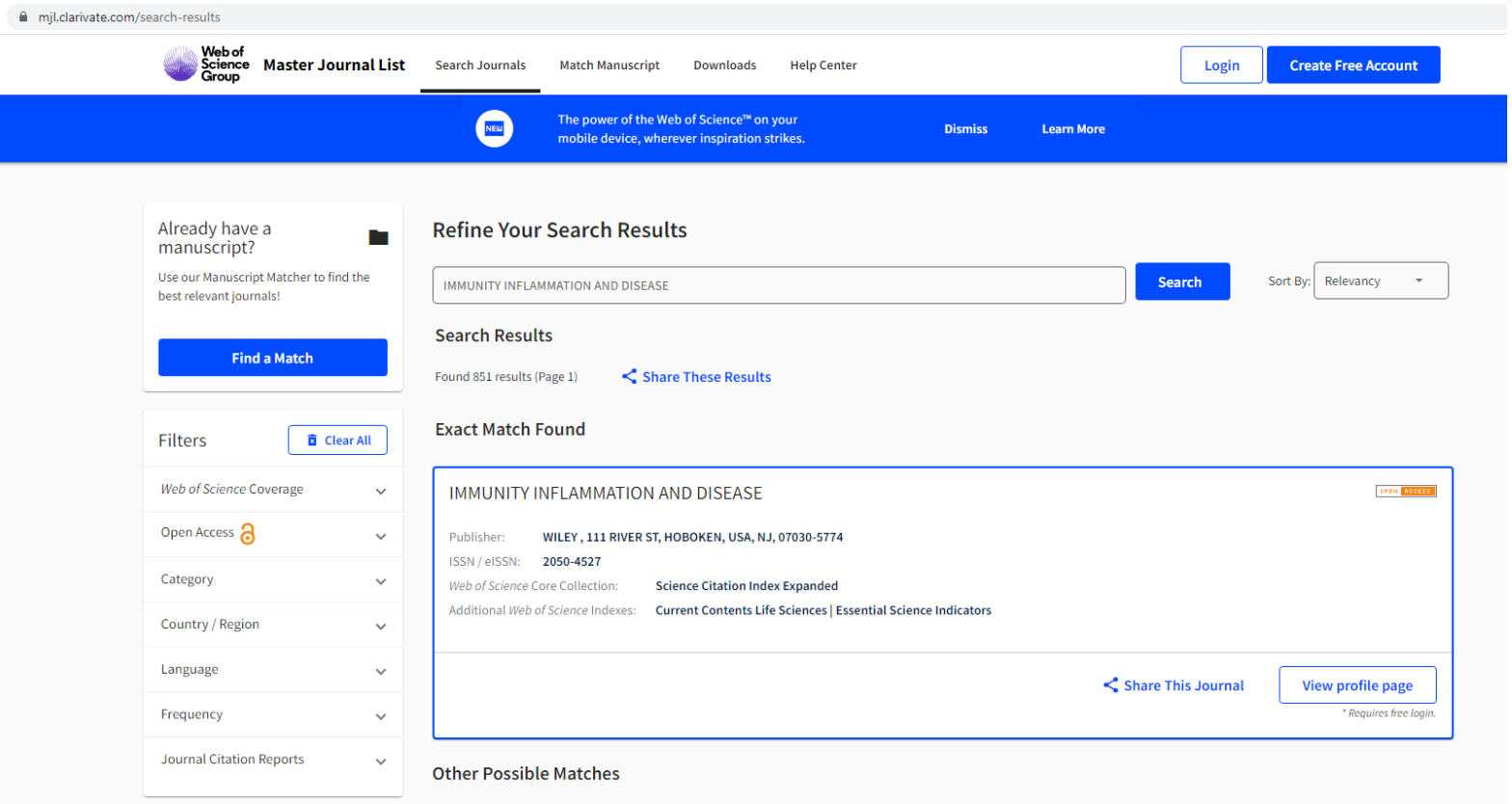
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