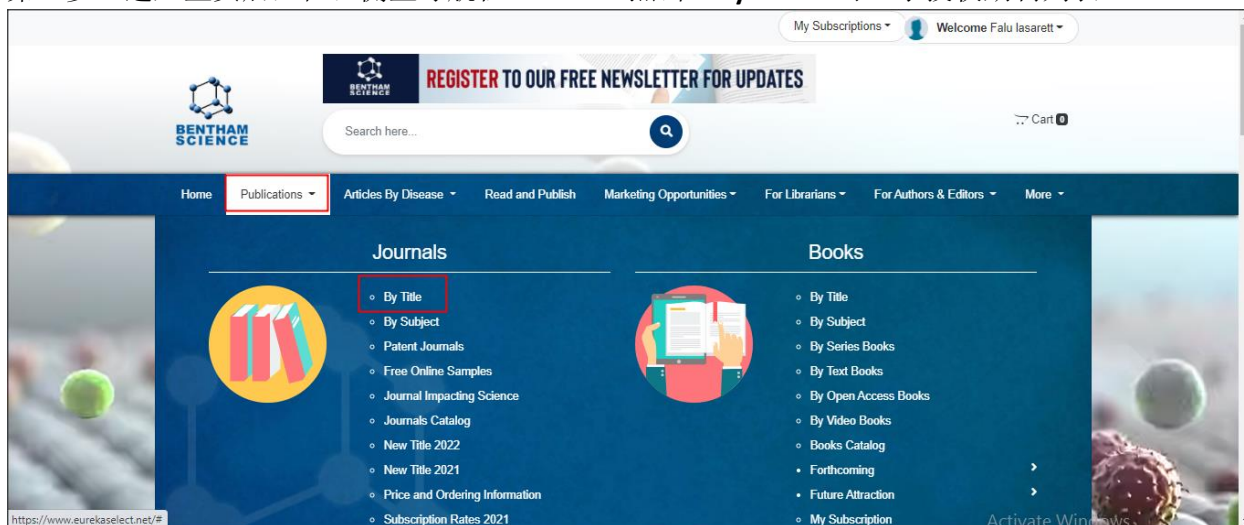


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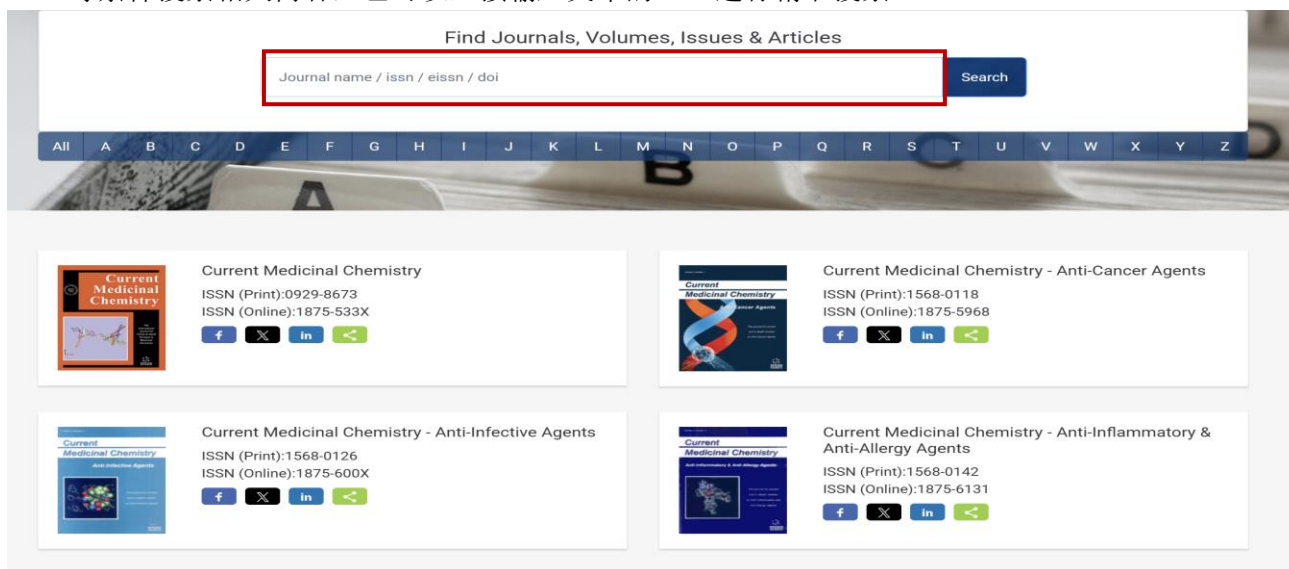
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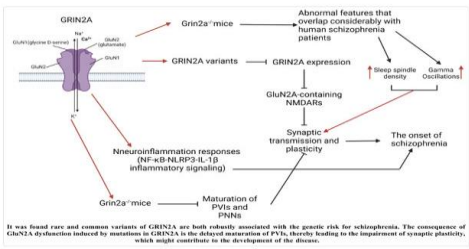
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Pp: 621-634
Author(s): Xiao-Ming Sheng and Wei Guan*
DOI: 10.2174/011570159X327712241023084944
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Abstract

Schizophrenia is a severe psychiatric disorder and a complex polygenic inherited disease that affects nearly 1% of the global population. Although considerable progress has been made over the past 10 years in the treatment of schizophrenia, antipsychotics are not universally effective and may have serious side effects. The hypofunction of glutamate NMDA receptors (NMDARs) in GABAergic interneurons has long been postulated to be the principal pathophysiology of schizophrenia. A recent study has shown that *GRIN2A* pathogenic variants are closely related to the aetiology of the disorder. *GRIN2A* encodes the GluN2A protein, which is a subunit of NMDAR. Most *GRIN2A* variants have been predicted to cause protein truncation, which results in reduced gene expression. Preclinical studies have indicated that *GRIN2A* mutations lead to NMDAR loss of function and substantially increase the risk of schizophrenia; however, their role in schizophrenia is not well understood. We hypothesise that the heterozygous loss of *GRIN2A* induces NMDAR hypofunction sufficient to confer a substantial risk of schizophrenia. Therefore, this review focuses on *GRIN2A* as a target for novel antipsychotics and discusses the mechanisms by which *GRIN2A* modulates antischizophrenic activities. Moreover, our review contributes to the understanding of the pathophysiology of schizophrenia to facilitate finding treatments for the cognitive and negative symptoms of schizophrenia.

Keywords: GRIN2A, Schizophrenia, NMDAR, pathophysiology, synaptic signalling, mutations.

Graphical Abstract



It was found rare and common variants of GRIN2A are both robustly associated with the genetic risk for schizophrenia. The consequence of GluN2A dysfunction induced by mutations in GRIN2A is the delayed maturation of PVs, thereby leading to the impairment of synaptic plasticity, which might contribute to the development of the disease.

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Pulmonary **Drug Delivery** System: A Novel Approach for **Drug Delivery**

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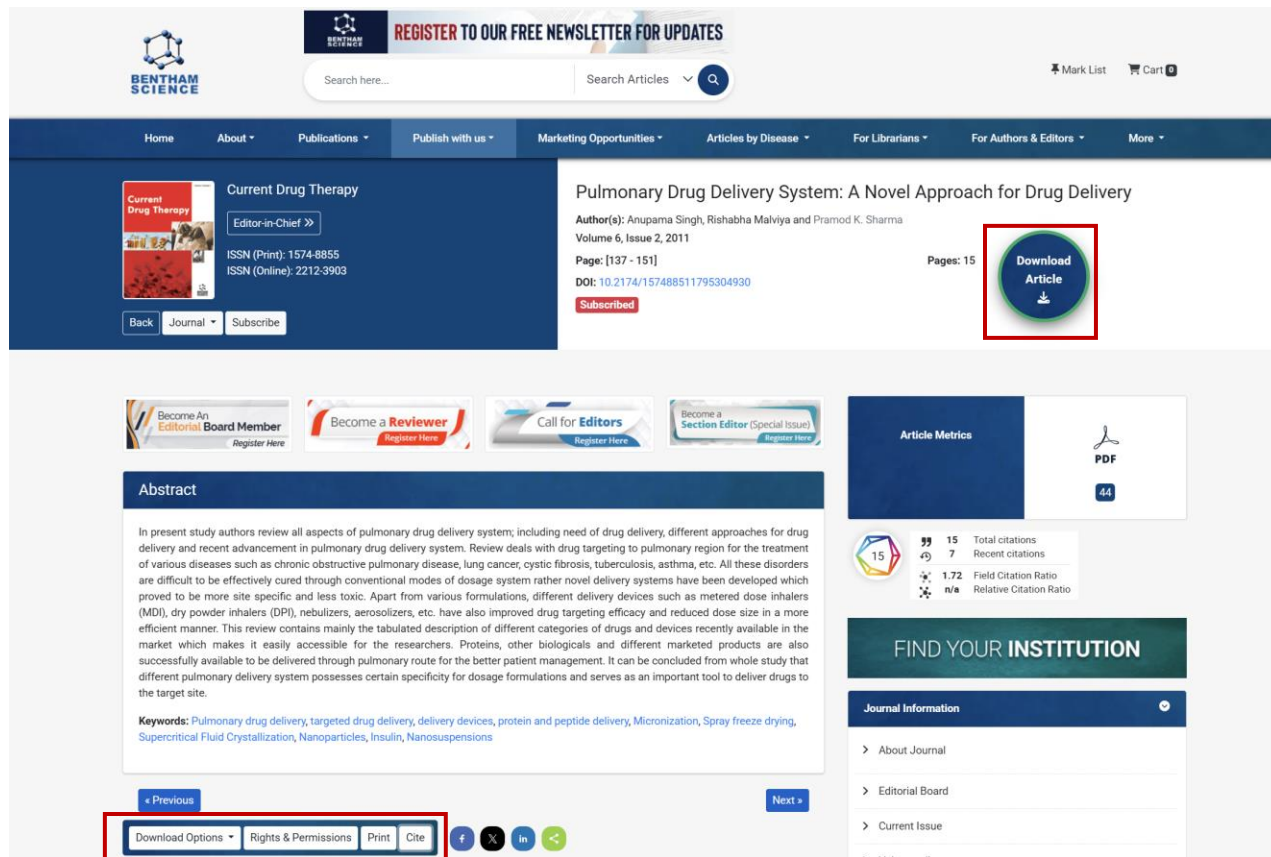
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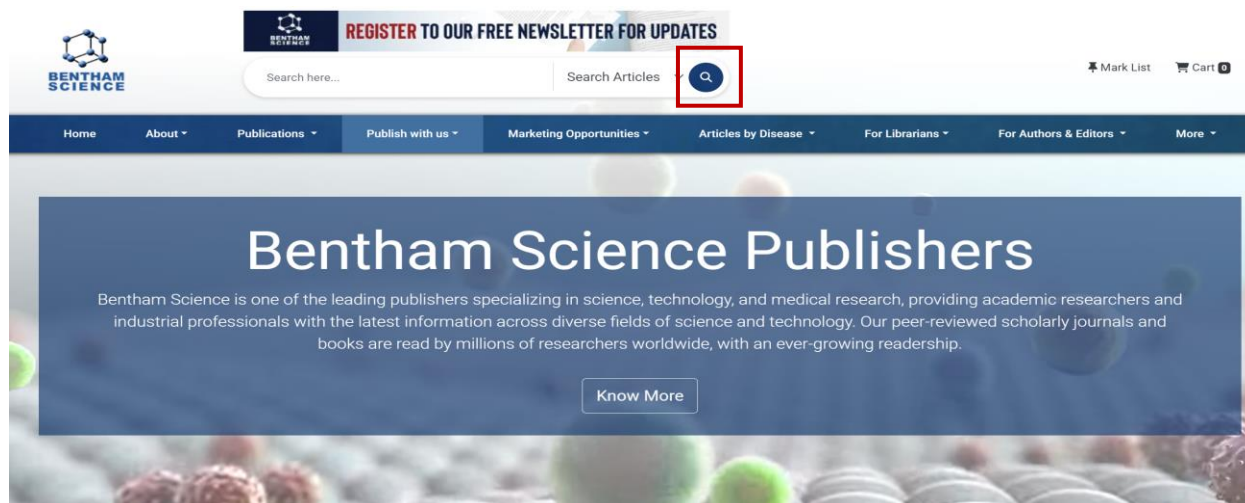
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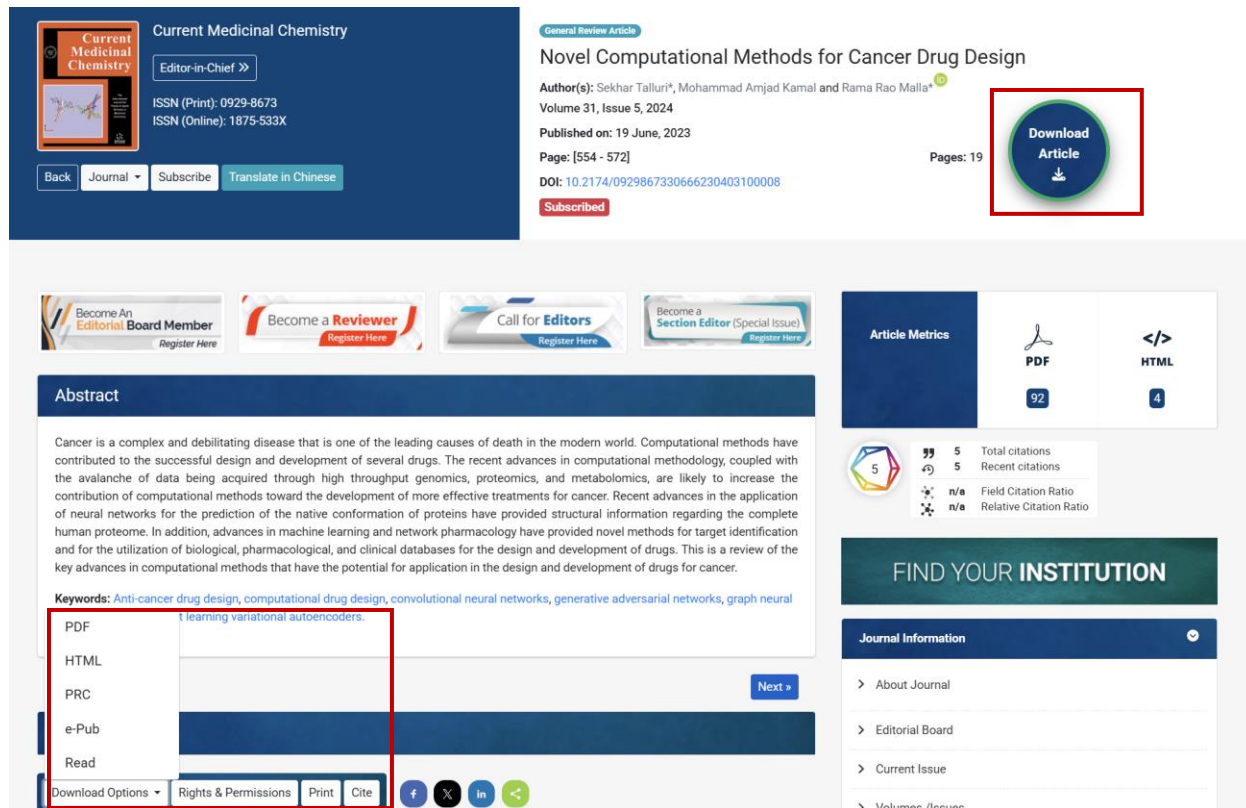
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Abstract
Cancer is a complex and debilitating disease that is one of the leading causes of death in the modern world. Computational methods have contributed to the successful design and development of several drugs. The recent advances in computational methodology, coupled with the avalanche of data being acquired through high throughput genomics, proteomics, and metabolomics, are likely to increase the contribution of computational methods toward the development of more effective treatments for cancer. Recent advances in the application of neural networks for the prediction of the native conformation of proteins have provided structural information regarding the complete human proteome. In addition, advances in machine learning and network pharmacology have provided novel methods for target identification and for the utilization of biological, pharmacological, and clinical databases for the design and development of drugs. This is a review of the key advances in computational methods that have the potential for application in the design and development of drugs for cancer.
Keywords: Anti-cancer drug design, computational drug design, convolutional neural networks, generative adversarial networks, graph neural network, machine learning, variational autoencoders.

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