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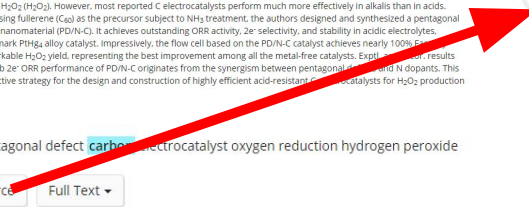
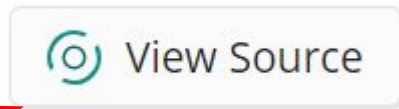


By: Zhang, Chang; Shen, Wangqiang; Guo, Kun; Xiong, Mo; Zhang, Jian; Lu, Xing

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Metal-free C-based materials are considered one of the most promising two-electron O₂ reduction reaction (2e⁻ ORR) electrocatalysts for the green synthesis of H₂O₂ (H₂O₂). However, most reported C electrocatalysts perform much more effectively in alkalies than in acids. Herein, by creatively using fullerene (C₆₀) as the precursor subject to NH₃ treatment, the authors designed and synthesized a pentagonal defect-rich N-doped C nanomaterial (PD/N-C). It achieves outstanding ORR activity, 2e⁻ selectivity, and stability in acidic electrolytes, surpassing the benchmark Pt/C alloy catalyst. Impressively, the flow cell based on the PD/N-C catalyst achieves nearly 100% Faraday efficiency with a remarkable H₂O₂ yield, representing the best improvement among all the metal-free catalysts. Experimental and theoretical results reveal that such superb 2e⁻ ORR performance of PD/N-C originates from the synergism between pentagonal defects and N dopants. This work presents an effective strategy for the design and construction of highly efficient acid-resistant C electrocatalysts for H₂O₂ production and beyond.

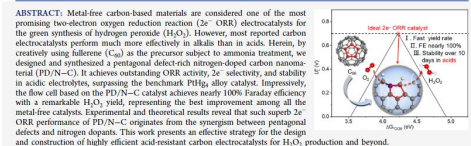
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A Pentagonal Defect-Rich Metal-Free Carbon Electro catalyst for Boosting Acidic O₂ Reduction to H₂O₂ Production

Chang Zhang, Wangqiang Shen, Kun Guo, Mo Xiong, Jian Zhang, and Xing Lu*

Cite This: *J. Am. Chem. Soc.* 2023, 145, 11589–11598



1. INTRODUCTION 2e⁻ selectivity in acidic electrolytes,^{23a–37} but they have



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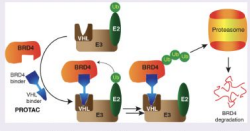
Selective Small Molecule Induced Degradation of the BET Bromodomain Protein BRD4

Michael Zengerle, Kwok-Ho Chan, and Alessio Ciulli*

College of Life Sciences, Division of Biological Chemistry and Drug Discovery, University of Dundee, James Black Centre, Dow Street, Dundee, DD1 5EH, United Kingdom

Supporting Information

ABSTRACT: The Bromo- and Extra-Terminal (BET) proteins BRD2, BRD3, and BRD4 play important roles in transcriptional regulation, epigenetics, and cancer and are the targets of pan-BET selective bromodomain inhibitor JQ1. However, the lack of intra-BET selectivity limits the scope of current inhibitors as probes for target validation and could lead to unwanted side effects or toxicity in a therapeutic setting. We designed Proteolysis Targeted Chimeras (PROTACs) that tether JQ1 to a ligand for the E3 ubiquitin ligase VHL, aimed at triggering the intracellular destruction of BET proteins. Compound MZ1 potently and rapidly induces reversible, long-lasting, and unexpectedly selective removal of BRD4 over BRD2 and BRD3. The activity of MZ1 is dependent on binding to VHL, but is achieved at a sufficiently low concentration not to induce stabilization of HIF-1 α . Gene expression profiles of selected cancer-related genes responsive to JQ1 reveal distinct and more limited transcriptional responses induced by MZ1, consistent with selective suppression of BRD4. Our discovery opens up new opportunities to elucidate the cellular phenotypes and therapeutic implications associated with selective targeting of BRD4.



The Bromo- and Extra-terminal (BET) family of proteins, including the ubiquitously expressed BRD2, BRD3, and BRD4 and the testis-specific BRDT, recruit transcriptional regulatory complexes to acetylated chromatin thereby controlling specific networks of genes involved in cellular proliferation and cell cycle progression.¹ Deregulation of BET protein activity, in particular BRD4, has been strongly linked to cancer and inflammatory diseases, making BET proteins attractive drug targets.² For example, RNAi screens have identified BRD4 as a therapeutic target in acute myeloid leukemias,³ ovarian carcinomas,⁴ and siRNA knock down of BRD4, but not of BRD2 or BRD3, induced upregulation of apolipoprotein A1 (ApoA1), which protects from atherosclerosis progression and other inflammatory processes.⁵ The silencing of BRD4 furthermore identified BRD4 as a target to treat chronic obstructive pulmonary disease (COPD).⁶ These results underscore the potential of targeting BRD4 as a therapeutic strategy and motivate further research in validating BRD4 as a drug target.

Crucial to the function of BET proteins are two highly homologous bromodomains that are present in their amino-terminal regions and direct recruitment to nucleosomes by binding to specific acetylated lysines (K_4) within histone tails. Small molecule BET inhibitors, including the triazolodiazepine-based JQ1⁷ and I-BET762⁸ (Figure 1a) among others,^{10–13} bind to the K_4 -binding pocket of the bromodomains and disrupt interaction with histones, thereby displacing BET proteins and their associated transcriptional regulatory complexes from chromatin. BET inhibitors are highly potent ($K_d \sim 100$ nM), cell-penetrant, and active *in vivo* against a range

of solid, hematological, and other tumors, which has prompted compounds entering phase I clinical trials for cancer.^{14–16} However, BET inhibitors show no selectivity for individual BET family members, thereby limiting their scope as chemical probes for validating the roles of individual BET targets in physiology and disease. To this end, chemical genetic strategies have been recently developed to engineer orthogonal, non-BET bromodomain-ligand pairs.¹⁷ While this approach has the advantage of enabling disruption at will of a single or more bromodomains, it requires a mutation to be introduced into the target protein. Therapeutically, the effects of BET inhibitors on different transcriptional pathways have raised concerns about the safety and tolerability of BET inhibitors in humans. Crucially, none of the inhibitors described to date is selective for binding BRD4 bromodomains over those of BRD2 and BRD3.

Small molecule chemical probes or inhibitors that act at the post-translational level hold several advantages over genetic techniques such as deletions or knockouts and RNAi knockdowns, affording spatial and temporal control in a reversible manner. A general limitation associated with conventional small molecule driven target inhibition is that it often demands full target engagement, requiring sustained high concentration of a potent

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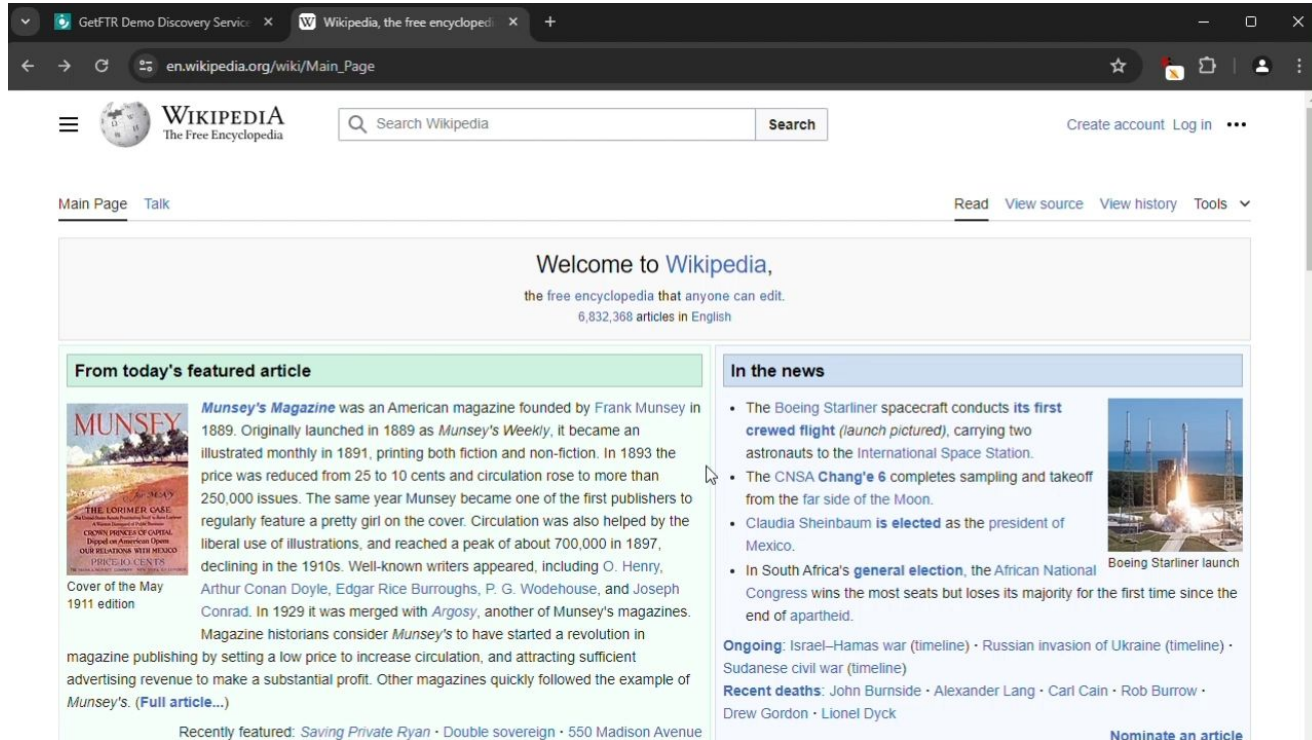
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From today's featured article

Munsey's Magazine was an American magazine founded by Frank Munsey in 1889. Originally launched in 1889 as *Munsey's Weekly*, it became an illustrated monthly in 1891, printing both fiction and non-fiction. In 1893 the price was reduced from 25 to 10 cents and circulation rose to more than 250,000 issues. The same year Munsey became one of the first publishers to regularly feature a pretty girl on the cover. Circulation was also helped by the liberal use of illustrations, and reached a peak of about 700,000 in 1897, declining in the 1910s. Well-known writers appeared, including O. Henry, Arthur Conan Doyle, Edgar Rice Burroughs, P. G. Wodehouse, and Joseph Conrad. In 1929 it was merged with *Argosy*, another of Munsey's magazines. Magazine historians consider *Munsey's* to have started a revolution in magazine publishing by setting a low price to increase circulation, and attracting sufficient advertising revenue to make a substantial profit. Other magazines quickly followed the example of *Munsey's*. ([Full article...](#))

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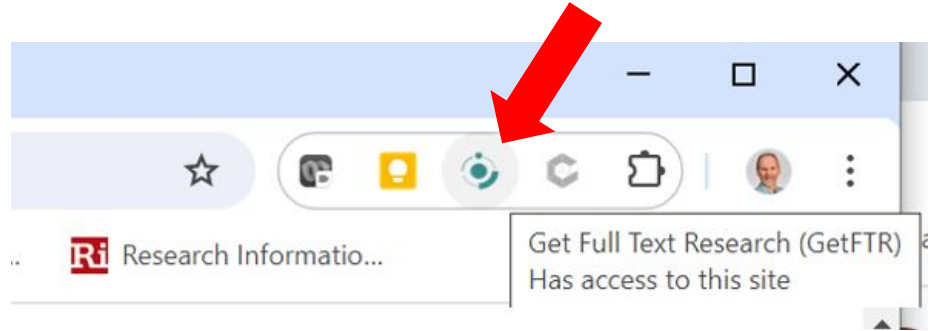
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