LETTER TO THE EDITOR

Letter to the editor: BET inhibitors might target innate inflammatory and profibrotic signaling networks in COPD

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TO THE EDITOR: Establishing new therapeutic approaches for patients with heart failure (HF) is of crucial importance. In a recent article from Science Translational Medicine, Duan et al. (1) demonstrated that bromodomain and extra-terminal motif (BET) bromodomain inhibitor JQ1 could treat HF by suppressing innate inflammatory and profibrotic transcriptional networks, which opened a new avenue for further development of BET inhibitors for HF. Since many other diseases such as chronic obstructive pulmonary disease (COPD) may share common pathological gene regulatory programs with HF, BET inhibitors might also have therapeutic effects in these diseases.

To validate this hypothesis, publicly available data from Gene Expression Omnibus (GSE47460) was downloaded and reanalyzed (2). First, differentially expressed genes in lung tissues from COPD (n = 145) compared with normal controls (n = 91) were computed, and a preranked gene list was built. Gene Set Enrichment Analysis (3) results indicated that COPD-related genes are positively correlated with NF-κB pathway activation, immune response, TGF-β pathway activation, and epithelial-mesenchymal transition (Fig. 1). This result indicates that COPD shares similar molecular patterns with HF. As was shown in the article by Duan et al. (1), innate inflammatory and profibrotic transcriptional networks could be suppressed by BET inhibitors. We believe BET inhibitors could also be worth trying in the treatment of COPD or other diseases such as liver cirrhosis, which may share the same pathological gene expression patterns.

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DISCLOSURES

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AUTHOR CONTRIBUTIONS

J.D. and Y.H. analyzed data; J.D. prepared figure; J.D. and Y.H. drafted manuscript; Y.H. conceived and designed research.

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Fig. 1. Chronic obstructive pulmonary disease gene expression signatures are positively correlated with NF-κB pathway activation (A), immune response (B), TGF-β pathway activation (C), and epithelial-mesenchymal transition (EMT; D). NES, normalized enrichment score.

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