Abundance of ACVR1B transcript is elevated during septic conditions: perspectives obtained from a hands-on reductionist investigation

JournalArticle (Originalarbeit in einer wissenschaftlichen Zeitschrift)

ID 4665583
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Year 2023

Title Abundance of ACVR1B transcript is elevated during septic conditions: perspectives obtained from a hands-on reductionist investigation

Journal Front Immunol

Volume 14

Pages / **Article-Number** 1072732

Mesh terms Humans; Melioidosis; Sepsis; Transforming Growth Factor beta, metabolism; Activin Receptors, Type I, metabolism

Sepsis is a complex heterogeneous condition, and the current lack of effective risk and outcome predictors hinders the improvement of its management. Using a reductionist approach leveraging publicly available transcriptomic data, we describe a knowledge gap for the role of ACVR1B (activin A receptor type 1B) in sepsis. ACVR1B, a member of the transforming growth factor-beta (TGF-beta) superfamily, was selected based on the following: 1) induction upon in vitro exposure of neutrophils from healthy subjects with the serum of septic patients (GSE49755), and 2) absence or minimal overlap between ACVR1B, sepsis, inflammation, or neutrophil in published literature. Moreover, ACVR1B expression is upregulated in septic melioidosis, a widespread cause of fatal sepsis in the tropics. Key biological concepts extracted from a series of PubMed queries established indirect links between ACVR1B and ”cancer”, ”TGF-beta superfamily”, ”cell proliferation”, ”inhibitors of activin”, and ”apoptosis”. We confirmed our observations by measuring ACVR1B transcript abundance in buffy coat samples obtained from healthy individuals (n=3) exposed to septic plasma (n = 26 melioidosis sepsis cases)ex vivo. Based on our re-investigation of publicly available transcriptomic data and newly generated ex vivo data, we provide perspective on the role of ACVR1B during sepsis. Additional experiments for addressing this knowledge gap are discussed.

ISSN/ISBN 1664-3224 (Electronic)1664-3224 (Linking)

URL https://doi.org/10.3389/fimmu.2023.1072732

edoc-URL https://edoc.unibas.ch/94563/

Full Text on edoc Available;

Digital Object Identifier DOI 10.3389/fimmu.2023.1072732

PubMed ID http://www.ncbi.nlm.nih.gov/pubmed/37020544

ISI-Number WOS:000961681900001

Document type (ISI) Journal Article