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Gene expression profile of Neutrophil Extracellular Traps (NETs) stimulated by L-amino acid oxidase from *Calloselasma rhodostoma* venom

Mauro Valentino Paloschi[1,*], Suzanne Nery Serrath[1,2], Milena Daniela Souza Silva[1,2], Daniel Gomes Cardozo[1], Andrey Victor Echeverria Ugarte[1], Charles Nunes Boeno[1], Jéssica Amaral Lopes[1], Cristina Matiele Alves Rego[1], Hallison Mota Santana[1], Yoda Janaina Ikenohuchi[1,2], Braz Junior Campos Farias[1], Kátia Paula Felipin[1,2], Micaela de Melo Cordeiro Eulálio[1,2], Larissa Faustina Cruz[1], João Gabriel dos Santos Magalhães[1], Sulamita da Silva Setúbal[1], Andreimar Martins Soares[2,3], Juliana Pavan Zuliani[1,2]

[1] Laboratório de Imunologia Celular Aplicada à Saúde, Fundação Oswaldo Cruz, FIOCRUZ Rondônia, Porto Velho-RO, Brazil.
[2] Programa de Pós-Graduação em Biologia Experimental, Universidade Federal de Rondônia - UNIR, Fundação Oswaldo Cruz, FIOCRUZ Rondônia, Porto Velho-RO, Brazil.
[3] Laboratório de Biotecnologia de Proteínas e Compostos Bioativos Aplicados à Saúde (LABIOPROT), Fundação Oswaldo Cruz, FIOCRUZ Rondônia e Departamento de Medicina, Universidade Federal de Rondônia, UNIR, Porto Velho-RO, Brazil.
* Correspondence: mauro.paloschi@fiocruz.br; Tel.: +55 69 3219-6000.

Introduction and Objectives: The aim of this study is to investigate the gene expression associated with the formation of neutrophil extracellular traps (NETs) stimulated by L-amino acid oxidase from *Calloselasma rhodostoma* (Cr-LAAO). LAAOs present in snake venom have been demonstrated to activate human neutrophils, leading to the generation of reactive oxygen species (ROS), chemotaxis, phagocytosis, NETs and the release of pro-inflammatory cytokines. Neutrophils are recognized for releasing NETs as a defense mechanism against pathogens. **Methods:** Neutrophils were isolated from peripheral blood of humans and incubated with either RPMI (control) or Cr-LAAO (50 µg/mL) for 1 hour, allowing for gene expression profiling using the GeneChip Clariom S Array Human.

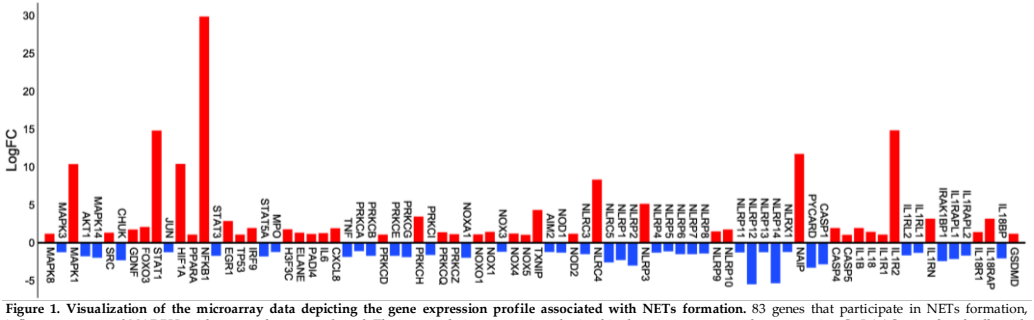

Figure 1. Visualization of the microarray data depicting the gene expression profile associated with NETs formation. 83 genes that participate in NETs formation, inflammasomes and NADPH oxidase complex were selected. The genes relative expression obtained in the microarray assay by com-paring Cr-LAAO-stimulated cells with negative control-stimulated (RPMI) cells were represent-ed by the fold change demonstrating the gene upregulation (red) and downregulation (blue) fold change bar plot.

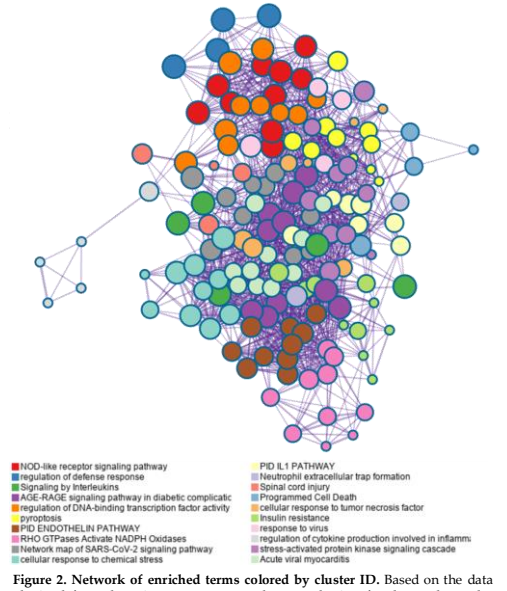

Figure 2. Network of enriched terms colored by cluster ID. Based on the data obtained from the microarray assay and gene selection for the study, nodes sharing the same cluster ID are typically located in close proximity to each other.

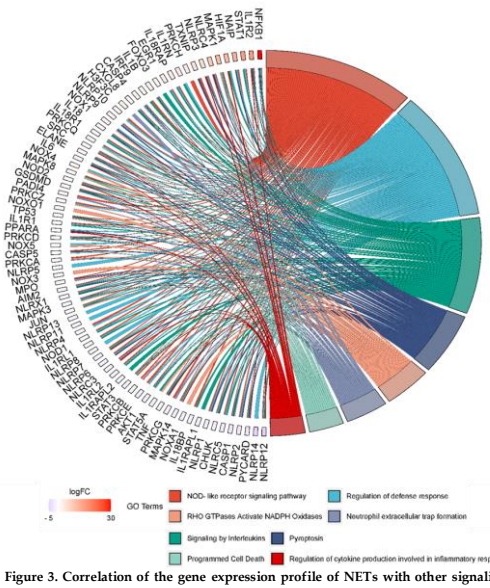

Figure 3. Correlation of the gene expression profile of NETs with other signaling pathways. Enriched items from pathways were compared with pathways related to the inflammatory process and plotted in a chord diagram.

Conclusion: This study provides valuable insights into the changes in gene expression caused by Cr-LAAO stimulation in human neutrophils. These findings align with earlier investigations and lay the groundwork for further exploration of the mechanisms involved in the inflammatory response triggered by Cr-LAAO.

References: doi: 10.1016/j.lfs.2022.120962; 10.1016/j.toxicon.2018.02.046; 10.1016/j.toxicon.2016.05.013; 10.1016/j.toxicon.2013.12.013.

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