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promoting the synthesis and local activation of MMPs, facilitating ECM-degradation as well as tumor aggressiveness and progression.

HEMATOLOGÍA

134. (097) **TRANSCRIPTIONAL AND PROTEOMIC ANALYSES OF REDOX ENVIOREMENT IN BETA-THALASSEMIA TRAIT**

Magdalena María Terán[1], María Eugenia Mónaco[2], Sineli Pedro Eugenio[3], Cecilia Haro[1], Sandra Stella Lazarte[1], Emilse Ledesma Achem[1], Natalia Álvarez Asensio[1], Ana Carolina Agüero Aguilera[1], Blanca Alicia de los Ángeles Issé[1]

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β-Thalassemia trait (BTT) is a heterogeneous group of genetic defects leading to decreased β -globin production, ineffective erythropoiesis and oxidative stress. Reactive oxygen species production and oxidative environment have an impact on all blood cell lineages. The aim was to evaluate, at transcriptional and proteomic levels, the pro-oxidative and anti-oxidative status in BTT patients. A descriptive study was performed with 66 subjects (40 apparently healthy and 26 with BTT). Real-time PCR was used for gene expression analyses of transcription factors forkhead homeobox typeO (FOXO3a) and nuclear factor erythroid2-related factor-2 (NRF2); antioxidant enzymes: catalase (CAT), peroxiredoxin-2 (PRX-2), superoxide dismutase (SOD); and cytokines TNF-α, IL-6. Quantitative mass spectrometry was performed on cytosol erythrocyte membranes depleted of hemoglobin. Bioinformatic analysis was performed with Perseus, BlastKoala and Proteome Discoverer V1.4 programs.
Relative expression of NRF2 was 4.7-fold higher in BTT than in control group, whereas FOXO3a expression was similar in both. Transcriptional expression of SOD, PRDX2 and proinflammatory cytokines were significantly upregulated in BTT compared to controls (p<0,005). Proteomic study showed significant difference in abundances of oxidative stress and inflammation markers such as lipoxygenase15 (ALOX15), poly-C-binding protein 1/2 (PCBP 1/2), P40/P67 subunit of NADPH oxidase and 70 kilodalton heat shock protein (HSP70), tyrosine-protein kinase (SYK) in BTT (p<0, 05). Proteins involved in redox imbalance protection such as glutathione S-transferase kappa1 (GSTκ1), isocitrate dehydrogenase 1/2 (IDH1/2) and glucose-6-phosphate dehydrogenase (G6PD) were higher in BTT than in controls (4.2, 4.1 and 2.1 fold respectively). These results showed changes in the gene expression of some redox regulators together with modifications in the erythrocyte proteome generated by the global redox imbalance underlying in this pathology.

135. (167) **REDOX AND INFLAMMATORY IMBALANCE IN PATIENTS WITH DEBUT OF ACUTE LEUKEMIA**

Ana Agüero Aguilera[1], Sandra Lazarte[1], María Eugenia Mónaco[2], Emilse Ledesma Achem[1], Blanca Issé[1], Magdalena Terán[1], Cecilia Haro[1]

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Several studies have reported the oxidative stress influence on the pathogenesis and evolution of hematologic neoplasms. The aim of this work was determine the behavior of redox and inflammatory biomarkers at transcriptional and systemic level in acute leukemia (AL) patients at the time of onset. Between 2016–2021, 61 AL patients and 63 controls (C) were evaluated. AL characterization was performed by blood count, cytochemistry and flow cytometry. Antioxidant enzymes *catalase* (CAT), and *superoxide dismutase* (SOD), and the cytokines *tumor necrosis factor-alpha* (TNF-α) and *interleukine-6* (IL-6) gene expression were analyzed with qPCR in peripheral white blood cells. Malondialdehyde (MDA) levels and CAT and SOD enzymatic activity were determined in serum by spectrophotometric methods. Cytokines concentration were measured by Human TNF-α and Human IL-6 ELISA. Statistical analyses were performed by SPSS V.25 statistical software and were considered significant at p<0,05. We detected 41% of acute lymphoid leukemia (ALL), 44% acute myeloid leukemia (AML) and 15% acute promyelocytic leukemia (APL). A significant increased level of lipoperoxidation was found in AML and APL respect to C [MDA μmol/L: AML=1,07 (0,40–6,60); ALP=1,22 (0,54–2,96); C=0,83 (0,33–2,51)]. Also, AML and APL showed higher CAT activity than C [CAT nmol/mg prot: AML=0,47 (0,02–3,62); ALP=0,70 (0,18–0,99); C=0,25 (0,08–2,15)], while SOD activity was a similar behavior between the groups studied. Furthermore, IL-6 concentration was significantly increased in all AL patients respect to C. SOD and IL-6 transcriptional expression were significantly downregulated in AL patients. No statistically significant differences were found in the other genes expression studied. These findings show the imbalance of redox and inflammatory biomarkers in the different AL evaluated and highlight on the differential behavior observed at the transcriptional/systemic level underlying in this neoplasm.

136. (288) **REGULATION OF HEPCIDIN BY ERYTHROPOIETIN IN MACROPHAGES**

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The role of erythropoietin (Epo) as a growth factor in erythropoiesis is well known. Many defects, both congenital and acquired, lead to ineffective erythropoiesis, thus establishing the condition of anemia. The demand for iron (Fe), necessary in biological processes, including erythropoiesis, gives importance to its controlled regulation. Hepcidin (Hep) is a key regulator of systemic iron metabolism and its expression responds to Fe levels. The participation of Epo in the regulation of Hep has been investigated in hepatocytes but not in macrophages, fundamental cells in Fe homeostasis that act as reservoirs of senescent erythrocytes. Therefore, the aim of this work was to investigate the regulation of Hep by Epo in macrophages.
Standardization of the differentiation of monocytic THP-1 cells to macrophages was carried out with different concentrations of phorbol 12-myristate-13-acetate (PMA). According to cell morphology (visible light microscopy), results of viability, proliferation and mRNA levels of the specific differentiation markers CD-14 and CD-68 (RT-PCR), 100 nM was chosen as the adequate PMA concentration to continue with the stated objective. The presence of EpoR was demonstrated by Western blotting (anti-EpoR M20), RT-PCR and flow cytometry, comparing with UT-7 cells as a positive control. Epo treatment of macrophages induced a significant decrease in Hep mRNA levels (RT-PCR, a.u.: C6h 0.39±0.02; *Epo6h 0.11±0.03; *Epo24h 0.09±0.04, *P<0.05 vs. C6h, n=5). To investigate whether this action of Epo takes place via its traditional pathway, evaluation of Hep levels was carried out in the presence of Jak2 (AG490) and PI3K (Ly294002) inhibitors and, under these conditions, Hep expression was found increased. In conclusion, the results show that Hep expression is modulated by Epo in macrophages, an effect that occurs through its receptor and is mediated by the Jak2/PI3K pathway.

137. (306) **ACTIVATION OF TOLL-LIKE RECEPTORS 7 AND 8 ON CD34+ CELLS BY COXSACKIEVIRUS B3 IMPAIRS MEGAKARYOCYTE AND PLATELET PRODUCTION**

Camila Sofía Rodríguez[1], Nancy Lorena Charó[1], Paula Oneto[1], Ricardo Gómez[2], Lina Paola D'Atri[1] y Mirta Schattner[1]

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Background: Increasing evidence indicates that hematopoietic progenitor cells (CD34+ cells), megakaryocytes (MKs), and platelets (PLT) express toll-like receptors (TLR) allowing the contribution of these cells to the immune response and inflammation. However,