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European Journal of Clinical Investigation

THE JOURNAL OF THE EUROPEAN SOCIETY FOR CLINICAL INVESTIGATION

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Conclusions: Structural and functional alterations occur in microsomes, mitochondria, and MAM fractions in AD cellular model. In this way, these results allow not only to clarify the pathophysiology of AD but may also be useful to devise a treatment aimed at restoring the normal MAMs function rather than dealing with the plaques and tangles accumulation, which are considered to be downstream consequences of alteration on MAM function and ER-mitochondrial connectivity.

55ASM-0122 ST | Mitochondria as a diagnostic marker between schizophrenia and bipolar disorder. Role of the interactome

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Background: Schizophrenia and bipolar disorder, with a prevalence of about 1% in the general population, have a prominent role among mental illnesses. The clinical and prognostic implications of the misdiagnosis rates between schizophrenic (SCH) and bipolar (BD) disorders and, consequently, the introduction of non-specific treatments force researchers to look for valid and reliable objective markers potentially useful to differentiate these disorders from the onset of the disease. Being the interactome the set of relationships that molecules establish within a cell, our intention was to describe differential interactomes between schizophrenia and bipolarity in peripheral blood mononuclear cells. These different molecules provide us with diagnostic discrimination markers. Thus, based on the symptomatology of both diseases, the discrepancy in their energetic capacity seemed plausible, we started the study at the mitochondrial level.

Materials and Methods: Peripheral blood mononuclear cells for mitochondrial characterization were isolated from 50 individuals without significant differences in gender. 22 SCH, 14 BD and the rest (14) healthy control individuals.

Results: The results showed a higher expression of mitochondrial complexes, higher mitochondrial dynamics and

higher ATP production in schizophrenic individuals compared to bipolar ones.

Conclusions: These different results obtained strongly support the possibility that mitochondrial functionality could be considered as a robust marker and reinforce the biological diagnostic importance to assist in the categorical discrimination among mental disorders.

J.C.B-M is a FISS pre-doctoral fellow (FI18/00149), J B and P G-P are members of “Centro de Investigación Biomédica en Red de Salud Mental (CIBERSAM)”

55ASM-0132 ST | Manipulation of glucose availability promotes different in vitro redox environments in human dermal fibroblasts

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Background: Changes in redox homeostasis are associated with the progression of age-related diseases, which may be prevented with antioxidants. However, clinical trials have been disappointing despite promising *in vitro* assays, suggesting that preclinical assays should be better designed to be more consistent with *in vivo* results. Since conventional cell culture assays introduce artifacts influencing redox research, and mitochondria are active players in cellular redox homeostasis, we aim to evaluate redox responses under different metabolic contexts by modulating cellular reliance on mitochondrial energy production *in vitro*.

Materials and Methods: Normal Human Dermal Fibroblasts (NHDF) were cultured in the absence of glucose (to stimulate oxidative phosphorylation, OXPHOS, OX medium) and compared with cells cultured in high glucose (HG) or low glucose (LG) media, to determine how different metabolic contexts can influence the cellular effects of oxidants, specifically H₂O₂ and *tert*-butyl hydroperoxide (*t*-BHP). Oxygen consumption rate (OCR) and extracellular acidification rate (ECAR) were measured using the Seahorse XFe96 Extracellular Flux Analyzer, mitochondrial network morphology was assessed by confocal microscopy, and substrate oxidation was determined using Biolog Metabolic Phenotype Microarrays. The response to oxidative stress was evaluated by resazurin and DCF fluorescence.

Results: NHDF cultured in OXPHOS conditions presented higher sensitivity to mitochondrial inhibitors, rotenone,